A DISSERTATION ON

INTUSSUSCEPTION

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CERTIFICATE

This is to certify that this dissertation entitled "**INTUSSUSCEPTION"** submitted is the original work done by **Dr. RAMASAMY. L,** Postgraduate in the Department of General Surgery, Madras Medical College and Government General Hospital Chennai in partial fulfilment of the requirement for the award of the Degree of M.S. General Surgery, September 2006 under my guidance and supervision. This dissertation is original and no part of this study has been submitted for the award of any other degree or diploma.

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DECLARATION

I solemnly declare that the dissertation titled "INTUSSUSCEPTION" is done by me at Madras Medical College & Govt. General Hospital, Chennai during the year of 2004 under the guidance and supervision of Prof. J. Ravishankar M.S.

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfilment of requirements for the award of M.S. Degree (Branch I) in General Surgery.

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Contents

Introduction

Intussusception is the invagination or telescoping of one portion of the intestine into the lumen of an immediately adjoining part. Invariably it is the proximal into the distal bowel. The typical case of intussusception is a well nourished male child of 4 – 12 month of age who is awakened from sleep with what seems to be violent abdominal pain. The child cries out and vomits and soon thereafter passes a normal stool. The patient seems to recover immediately and may resume normal eating habits until stricken by another bout of colicky pain. With these spasms of pain he draws his knees up onto the abdomen. During each bout of pain he turns pale and may sweat and become markedly apathetic and lethargic, a sign that is now well-recognized. Thereafter he vomits repeatedly, has obvious bouts of recurrent peristaltic pain and begins to pass bloody mucus per rectum. Persistent apathy, pallor and evidence of dehydration are common signs. The intestinal obstruction, which has been present from the onset of symptoms gradually, becomes clinically manifest. The condition is likely to be fatal in 2-5 days if not corrected. The invagination of the intestine usually begins in the distal ileum or at the ileocaecal valve. Examination of the intestine shows no

obvious local anatomical cause for intussusception in most infants. Although the classic picture is typical, failure to recognize the occasional non-classical presentations may result in a delay in the diagnosis and an adverse outcome.

Infants with untreated intussusception often die from hypovolemia and the concurrent intestinal obstruction. A rare occurrence is for the area of intussusception to become gangrenous and slough, with the intestine fusing at the entrance of the intussusception and preserving intestinal continuity, while the necrosed intussusception is passed per rectum. Recovery may be permanent or stricture may occur, at the site of autoanastomosis.

Post operative intussusception has occurred after a wide variety of surgical procedures and is often difficult to diagnose because the symptoms are often obscure. The possibility should be kept in mind when a child develops sudden early signs of obstruction after a routine laparotomy.

Aims and Objectives

1. To study the incidence of intussusception among children and adults and the varied clinical presentation of intussusception in Government General Hospital, Chennai and in the Institute of Child Health, Egmore with its attached paediatric surgery department, Chennai.
2. To study the age incidence, sex incidence and the etiology of intussusception.
3. To study the treatment modalities according to the time taken for the diagnosis.
4. To emphasize the need for quick and proper decision in diagnosis, management and outcome.
5. To review the literature on the subject.

Materials and Methods

This study was conducted in the Department of General Surgery, Government General Hospital, Chennai and in the Department of Pediatric Surgery, Institute of Child Health, Egmore, Chennai during the period January 2004 to December 2004.

All cases admitted in the ward with the provisional diagnosis of intussusception have been taken for this study and followed up with the investigations. Confirmed cases of intussusception were only taken up for study and evaluation done. The following proforma was followed in evaluating all these patients.

Proforma

Clinico-pathological study of intussusception among children and adults

Name: age: Sex: IP no:

S.no: Address: Unit:

Socio-economical status: Occupation:

DOA: DOS: DOD:

Presenting complaints:

Abdominal pain:
- Location
- Duration
- Nature
- Progression
- Aggravating / relieving factors

Vomiting:
- Duration
- Contents
- Bile stained / non-bile stained

Bleeding PR:
- Duration
- Frequency
- Amount
- Colour
- Fresh / altered

Abdominal distension:
- Location
- Duration
- Progression
- Reduces after defecation / flatus

Mass abdomen:
- Duration
- Location
- Progression
- Associated with pain

Bowel habits:

- Diarrhoea
- Constipation
- Obstipation
- Mucus in stools
- Red currant jelly stools

Fever:

- Duration
- Low grade / high grade
- Progression
- Continuous / Intermittent

Nausea:

Anorexia:

Loss of weight

Past history

- Other medical illness
- H/o medication
- H/o similar episodes

Personal history

- Diet: vegetarian / non-vegetarian
- Smoking
- Alcoholism

Family history

Menstrual history

Treatment history

Antenatal history

Developmental history

Immunization history

General examination

Nutritional status
Dehydration
Pallor
Icterus
Generalised lymphadenopathy

Vitals

Pulse, BP, Temperature, RR

CVS

RS

Abdomen

Inspection:

VGP/VIP
Visible mass
Abdominal distension

Palpation:

Tenderness
Guarding
Rigidity
Rebound tenderness
Palpable mass

Percussion:

Any free fluid

Auscultation:

Bowel sounds

DRE

Mass
Bleeding PR
Tenderness

Clinical diagnosis

Investigations

Urine:

Alb / sugar/Dep

Blood:

- Complete haemogram
- Blood urea, sugar, and serum creatinine
- Serum electrolytes

X ray chest PA view

Plain x ray abdomen erect / supine view

USG abdomen

CT scan abdomen

Barium enema

Management

Non-Surgical:

- Hydrostatic reduction
- Pneumatic reduction

Surgical:

- Manual reduction
- Resection and anastomosis
- Colostomy or ileostomy

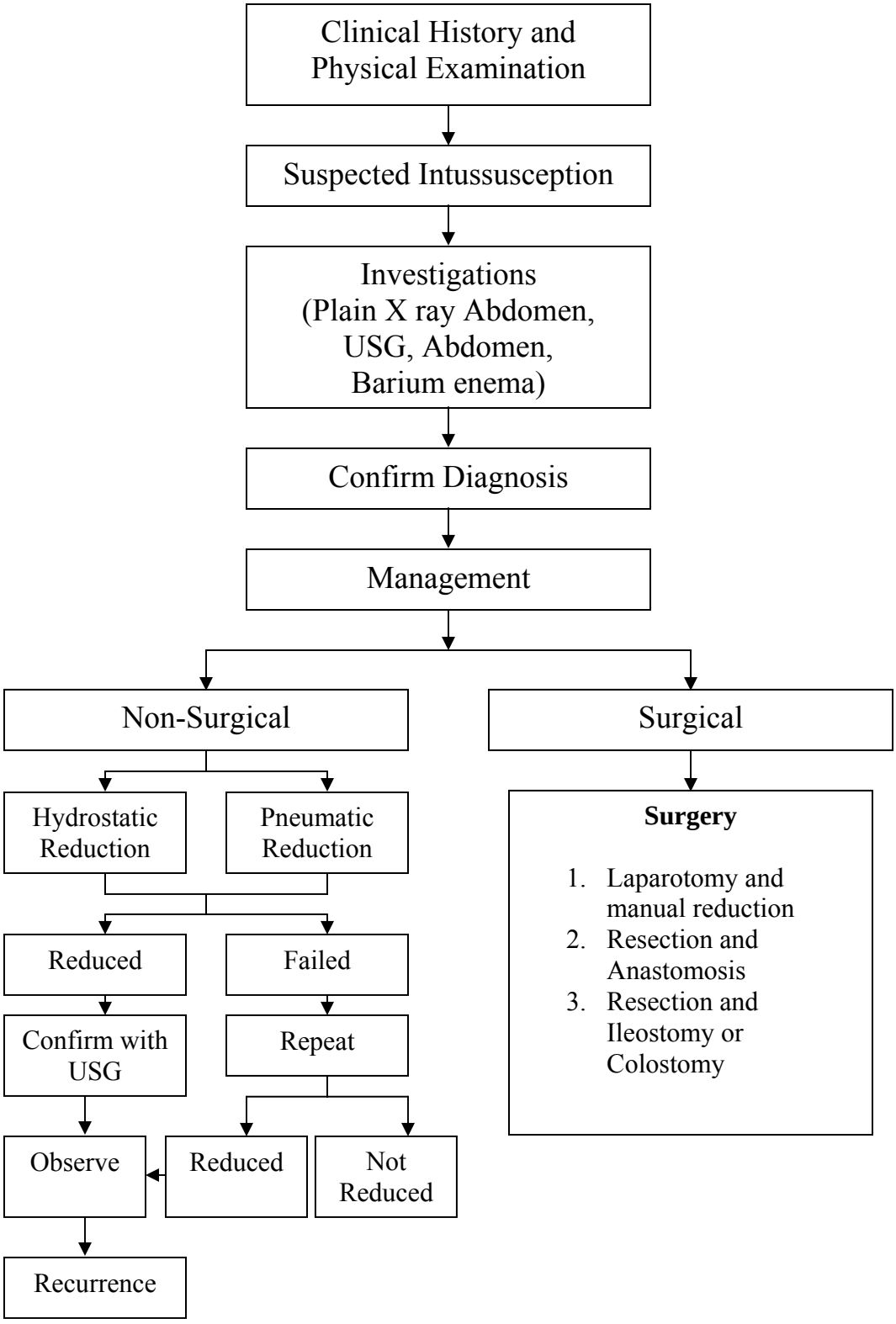
Post op complications

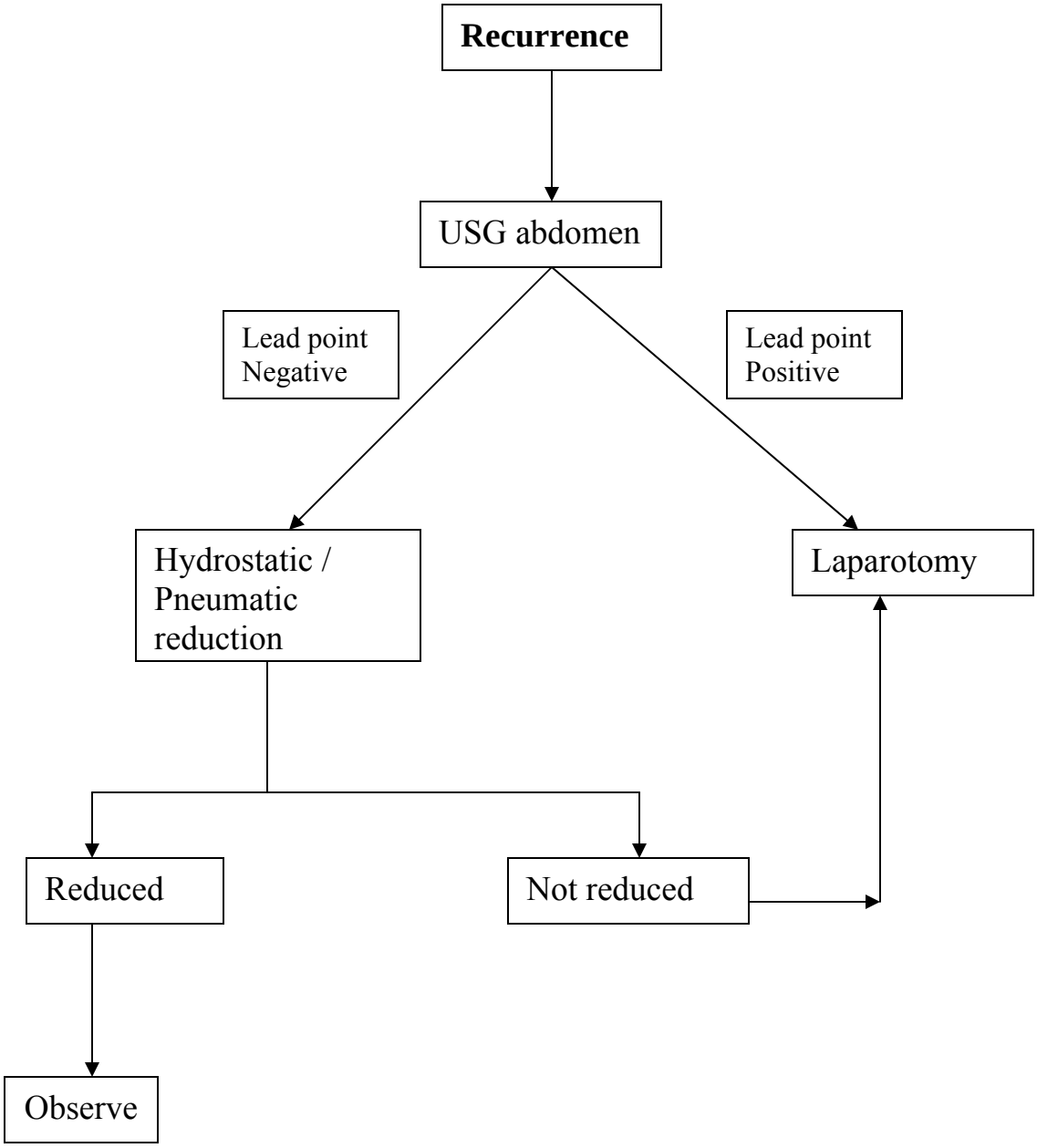
Histo-pathological examination

Follow up

- Observation
- Recurrence
- Reintervention

Treatment Protocol




Recurrence
USG abdomen
Lead point Negative
Lead point Positive
Hydrostatic / Pneumatic reduction
Laparotomy
Reduced
Not reduced
Observe

REVIEW OF LITERATURE

Development of Gut

Just before the 6th week of intrauterine life, the alimentary canal is a simple tube suspended in the midline of the abdominal cavity by a ventral and dorsal mesentery passing through to the hindgut. By the end of the 6th week, due to the development of the liver, rotation of the midgut loop occurs.

First stage of rotation is complete when the midgut loop is rotated through 90º in an anticlockwise direction. Growth in the length of small bowel causes 180º of anticlockwise rotation before the return of the bowel to the abdomen.

Second stage of rotation occurs at 10th to 11th week. The gut being too bulky to be returned en masse, it retreats in a defined order. The pre-axial portion returns first. While the pre-axial segment is returning, the superior mesenteric artery is firmly fixed to the umbilicus by its termination.

- The duodenum crosses behind the upper part of the superior mesenteric artery
- Transverse colon cross in front of the upper part of the superior mesenteric artery
- Descending colon has been pushed into the left flank
- Caecum is in the right loin.
- The coils of the small bowel range from left upper to right lower segments of the abdomen

During the third stage of rotation, which occurs between the 11^{th} week and shortly after birth, Caecum descends further reaching the right iliac fossa. The mesentery of the small gut becomes adherent to the posterior abdominal wall. Certain parts of gut become fixed to the posterior abdominal wall by fusion of these primitive mesenteries, with the posterior parietal peritoneum. The post arterial mesentery of transverse colon persists as the transverse mesocolon. The mesentery of the Caecum, ascending colon, hepatic flexure and hindgut becomes completely obliterated by fusion with the posterior parietal peritoneum except in the case of pelvic colon where the mesentery persists as the future mesocolon. There are 3 gut arteries that leave the aorta and pass ventrally to supply the alimentary tract.

The most cranial artery passes in the dorsal mesogastrium to supply the foregut. The next passes though the dorsal mesentery to supply the midgut and the last passes through the dorsal mesocolon to supply the hindgut. They are the celiac, superior mesenteric and inferior mesenteric arteries respectively and they continue to supply the derivatives of these parts of the alimentary canal in the adult. As the midgut loop returns to the abdominal cavity, the hindgut swings on its dorsal mesocolon and the mesocolon fuses within the parietal peritoneum of the left paracolic gutter. Hence the left colic vessels lie in front of everything else on the posterior abdominal wall. At the pelvic brim, fusion of the layers is not complete and a small part of the intestinal edge of the dorsal mesocolon of the hindgut remains free as the sigmoid mesocolon of the adult.

Name	**Extent**	**Artery**	**Function**
Foregut	Stomach and duodenum as far as the entry of the bile duct	Coeliac Artery	Digestive
Midgut	From the ampulla of vater to the junction of middle with the left third of transverse colon	Superior Mesenteric Artery (SMA)	Absorptive
Hindgut	The left colon	Inferior Mesenteric Artery (IMA)	Excretory

Anatomy of Small Bowel

The small bowel consists of 3 parts: duodenum, jejunum and ileum.

Duodenum:

25 cm long, it makes a C-shaped band which embraces the head of the pancreas. It consists of four parts:

- *1st part* – 5 cm long, it extends up and backwards to the right, to the level of the upper border of L_1
- *2nd part* – 7.5 cm long, it extends downwards, to the level of the lower border of L_3
- *3rd part* – 10 cm long, it extends to the left and crosses in front of L_3
- *4th part* – 2.5 cm long, it extends upwards to the level of L_2

In general, the anterior surface of the duodenum is covered with peritoneum except where the transverse colon crosses the 2nd part and holds the peritoneum away. Posterior surface and concavity of the 'C' are devoid of peritoneum.

Jejunum and Ileum:

60% of the total length of the GIT is composed of jejunum and ileum. The length of the small bowel varies from 4 to 6 m and the upper 2/5th is the jejunum and the lower 3/5th is ileum. It is entirely surrounded by the peritoneum. The small gut is supplied by its mesentery which extends from the left side of the 2nd lumbar vertebra to the right iliac fossa crossing the 3rd part of the duodenum aorta and vena cava and the right ureter in its course. It is 1.5 m long along this line of attachment. But along its free border it is as long as the small gut. Its depth is 15 cm except in relation to the parts of the small gut whish occupy the pelvis where it is 20 cm.

Differences between Jejunum and Ileum:

Jejunum	**Ileum**
Thicker wall	Thinner wall
Larger lumen	Smaller lumen
Fat on mesentery	Fat on ileum and mesentery
Prominent plicae circulates	Prominent plicae
Single line of arterial arcades	Several lines of arterial arcades
High narrow windows	Long broad windows
Aggregates of lymph nodules are sparse	Aggregates of lymph nodules are frequent

Ileocaecal Valve:

First described by Baubin in 1579, the ileocaecal valve was considered as a slit like valve with two major lips. In 1914, Rutherford noticed the difference between the valve of cadaver and that of the living patient. The valve in most patients resembles the cervix protruding into the vagina or the pyloric opening into the duodenum. The slit like orifice of the ileocaecal valve appears to be a post mortem artifact. The valve changes its appearance from a papilla to a bilabial valve that takes place within the first hour after death. Two rings of the thickened circular muscle, one at the base of the papilla and one at the free end form the closing mechanism of papilla.

Meckel's Diverticulum:

The vitello intestinal duct closes at its umbilical end and remains open at the intestinal end. This is called as the Meckel's Diverticulum. Meckel (1781-1833) described this diverticulum iliei verum. It occurs in 2% of the people. The diverticulum is 5 cm (2 inches) long and is found 60 cm (2 feet) from the ileo caecal valve. In 2% of the cases accessory pancreatic tissue occurs in the vestige. It is a true diverticulum, which is attached to the anti-mesenteric border of ileum. It may or may not possess a mesentery. Its apex may be free or attached by a fibrous band to the umbilicus or to the

mesentery in which case it can cause intestinal obstruction. Heterotropic gastric mucosa may or may not occur at the neck of the diverticulum. A diverticulum with a broad base may cause intussusception[2]. If the diverticulum has a narrow neck, stasis of its contents may precipitate the development of Diverticulitis[2].

Caecum:

This blind pouch of the large intestine projects downwards from the commencement of ascending colon below the ileo caecal junction. It is usually completely covered by peritoneum. There are two peritoneal folds from either side of posterior wall of Caecum forming between them the retrocaecal recess in which the appendix may lie. As in the rest of the colon, the longitudinal muscle of caecum is concentrated into three flat bands – taenia coli, which lie one anterior, one postero-medial and one postero-lateral. All three converge at the base of the appendix. Internally the ileo caecal valve, whose almost transverse slips may help to prevent some reflux into the ileum, guards the ileo caecal junction. In infants the caecum is conical and the appendix extends downwards from its apex. The lateral wall outgrows the medial wall and bulges down below the base of the appendix in the adult. The base of appendix thus comes to lie in the posteromedial wall

of the caecum. The caecum lies on the peritoneal floor of the right iliac fossa over the iliacus and psoas fasciae and the femoral and lateral femoral cutaneous nerves. Its lower end lies at the pelvic brim.

Appendix:

The vermiform appendix is a blind ending tube varying in length from 6 to 20 cm, which opens into the postero-medial wall of the caecum 2 cm below the ileo caecal valve. While the position of its base is constant in relation to the caecum, the appendix itself may lie in a variety of positions. The most common being the retrocaecal position. The three taenia of the caecum merge into a complete longitudinal muscle layer for the appendix. The sub mucosa contains many lymphoid masses and the lumen us thereby irregularly narrowed. The lumen is wider in the young child and may be obliterated in old age.

Mesoappendix is a triangular fold of peritoneum from the inferior layer of mesentery of the terminal ileum. A small fold of peritoneum extends from the terminal ileum to the front of the mesoappendix. This is the ileo caecal fold or bloodless fold of Treves and the space between it and the mesoappendix is the inferior ileo caecal recess. Another fold lies in front of the terminal ileum between the base of mesentery and the anterior wall of

caecum. This fold is raised by the contained anterior caecal artery and is called the vascular fold of caecum. The space behind it is the superior ileo caecal recess.

Ascending Colon:

It is 15 cm long and extends from the ileo caecal junction to the hepatic flexure. The ascending colon lies on the iliac fascia and the anterior layer of lumbar fascia. Its front and both sides possess a serous coat, which run laterally into the paracolic gutter and medially into the right infra colic compartment. The original embryonic mesentery is retained in 10% of adults. The taenia coli lie anteriorly postero-lateral and postero-medially. The ascending colon is sacculated due to the three taenia being too short for the bowel. Small pouches of peritoneum distended with fat, the appendices epiploicae project in places from the serous coat.

Transverse Colon:

This part of the colon, 45 cm long, extends from the hepatic flexure to the splenic flexure in a loop, which hangs down to a variable degree between the two fixed points anterior to the coils of the jejunum and ileum. The transverse colon is completely invested in peritoneum. It hangs free on the

transverse mesocolon. The splenic flexure lies at a higher level than the hepatic flexure. Appendices epiploicae are larger and more numerous. The taenia lie posteriorly, antero-superiorly and antero-inferiorly.

Descending Colon:

The descending colon is 30 cm and extends from the splenic flexure to the pelvic brim. The descending colon lies on the lumbar and iliac fascia. It ends at the pelvic brim 5 cm above the inguinal ligament. It is plastered to the posterior abdominal wall by peritoneum, though a mesentery is present in about 20% of the adults. The taenia lies anteriorly, postero-medially and postero-laterally. Appendices epiploicae are numerous. A fold of peritoneum, the phrenico colic ligament attaches the splenic flexure to the diaphragm at the level of the 10^{th} and 11^{th} ribs.

Sigmoid Colon:

Formerly known as the pelvic colon, this extends from the descending colon at the pelvic brim to the commencement of the rectum in front of the 3^{rd} piece of sacrum. It is 45 cm long, completely invested in peritoneum and hangs free on a mesentery, the sigmoid mesocolon. The commencement of sigmoid mesocolon is sacculated by three taenia coli. These muscular bands

are wider than elsewhere in the large gut. The sigmoid colon possesses well-developed appendices epiploicae. It lies usually in the pelvic cavity, coiled in front of the rectum, lying on the peritoneal surface of the bladder and uterus.

Blood Supply

I. Superior Mesenteric Artery

This is the artery of midgut and it supplies the gut from the entrance of bile duct to a level just short of the splenic flexure of colon. This artery arises from the front of the aorta at the level of lower border of L_1 vertebra.

Superior Mesenteric Vein lies in its right side. With its vein it enters the upper end of mesentery of small intestine and passes down to the right along the rest of mesentery.

a. Inferior Pancreatico Duodenal Artery

This is the first branch of the superior mesenteric artery arising from the posterior surface. It may come off the first jejunal branch. It supplies both the duodenum and head of pancreas and anastomoses with the superior pancreatico duodenal artery

b. Jejunal and Ileal Branches

These branches arise from the left of main trunk, passes down between the two layers of mesentery. The jejunal branches join each other in a series of anastomosing loops to form arterial arcades – single from upper jejunum double lower down. From the arcades straight arteries pass to the mesenteric border of gut. These vessels are long and close together forming high narrow windows in the intestinal border of the mesentery.

The ileal arteries are similar but form a larger series of arcades 3-5, the distal most lying near the ileal wall, so that the straight vessels branching off the arcades are shorter. There is more fat in this part of mesentery. Hence, the windows are not seen.

Occlusion of straight artery may lead to infarction of the segment supplied because these are end arteries. But occlusion of arcade arteries is usually without effect due to their numerous anastomotic connections.

c. Ileo Colic Artery

This artery arises from the right side of the superior mesenteric artery and divides into the superior and inferior branches. The superior branch runs up along the left side of the ascending colon to anastomose with the right colic artery. The inferior branch runs to the ileo colic junction and gives off

the anterior and posterior caecal arteries, an appendicular artery and an ileal branch which ascends to the left of the ileum to anastomose with the terminal branch of superior mesenteric artery.

d. Right Colic Artery

Arises from the right side of the superior mesenteric artery or in common with the ileo colic artery, divides into two branches. The descending artery runs down to anastomose with the superior branch of the ileo colic artery. Ascending branch runs up to the hepatic flexure where it anastomoses with a branch of middle colic artery.

e. Middle Colic Artery

Arises from the right side of superior mesenteric artery and descends between the two leaves of the transverse mesocolon. It lies to the right of midline and divides into the right and left branches which run along the margin of the transverse colon. Right branch anastomoses with the ascending branch of right colic artery and the left branch supplies the transverse colon almost to the splenic flexure where it anastomoses with the branch of left colic artery.

II. Inferior Mesenteric Artery

This is the artery of the hindgut. It arises from the front of aorta behind the inferior border of 3rd part of duodenum opposite L_3 vertebra at the level of the umbilicus 3 or 4 cm above the aortic bifurcation. It is smaller than superior mesenteric artery. It crosses the pelvic brim at the bifurcation of left common iliac vessels over the sacro iliac joint, at which point it converges towards the ureter. It gives off the left colic and sigmoid arteries.

a. Left Colic Artery

It leaves the trunk and passes upwards to the left behind the peritoneum. It divides into ascending and descending branches. Ascending branch is crossed anteriorly by the inferior mesenteric vein. The branches of these two arteries anastomose with each other as well as (above) with the left branch of middle colic artery (below) with the highest sigmoid artery.

b. Sigmoid Artery

Sigmoid arteries are 2 to 4 branches that pass between the layers of sigmoid mesocolon in which they form anastomosing loops. The last sigmoid branch anastomoses with the first branch of superior rectal artery.

Venous Drainage

I. Midgut:

Each branch of superior mesenteric artery is accompanied by a vein. All these veins flow into the superior mesenteric vein, a large trunk which lies to the right of the artery. It crosses the 3rd part of the duodenum and the uncinate process of pancreas. Behind the neck of the pancreas, it is joined by the splenic vein to form the portal vein.

II. Hindgut

Superior rectal vein runs up in the root of the sigmoid mesocolon on the left of superior rectal artery to the pelvic brim, above which it is named as the inferior mesenteric vein. This receives tributaries identical with the branches of inferior mesenteric artery. The vein itself runs vertically upwards to the left of artery, beneath the peritoneal floor of left infra colic compartment. The inferior mesenteric vein passes behind the lower border of the body of the pancreas in front of the left renal vein and joins the splenic vein.

Lymph Drainage

From the whole length of GIT, the lymph vessels pass back along the arteries, to the lymph nodes that lie in front of the aorta at the origin of the gut arteries. These comprise the celiac, superior mesenteric and inferior mesenteric group of lymph nodes.

The first filtering mechanism consists of isolated lymphoid follicles, which lie, in the mucous membrane of the alimentary canal from mouth to the anus. These lymphoid follicles are more numerous in the small intestine. In the lower part of ileum they become aggregated into the anti mesenteric border of ileum and are oval in shape, with their long arteries lying longitudinally along the ileum. In the large intestine, the lymphoid follicles in the mucous membrane are numerous and isolated from each other.

Lymph vessels pass from the follicles in the mucous membrane through the muscular wall of the gut to nearby nodes. Small and large intestines have a common pattern of 3 group nodes. First group lies in the peritoneum adjacent to the margin of gut, the mural nodes in the mesentery of small intestine and the para-colic nodes of the large intestine. Second group of intermediate nodes lie along the main blood vessels of supply and the third group are the para-aortic nodes at the origin of celiac, the superior mesenteric and inferior mesenteric arteries. The large intestine has some

additional nodes that lie on the external surface of gut wall and occasionally in the appendices epiploicae. These are the epicolic nodes.

Nerve Supply

All parts of the gut and its derivatives are innervated by the sympathetic and para sympathetic nerves, which travel together along the gut arteries. The nerve cells and fibres that supply muscle, blood vessels and glands are concentrated in two plexuses. The myenteric plexus of Auerbach is situated between the two muscle layers of the gut and the submucous plexus of Meissner is in the submucosa. These plexuses form the enteric nervous system. The system receives post ganglionic sympathetic (inhibitory) and preganglionic parasympathetic (excitatory) fibres. Pain impulses are transmitted by sympathetic fibres while impulses mediating sensations of distension pass in the parasympathetic fibres.

Physiology

Small Intestine

In the small intestine, intestinal contents are mixed with the secretion of the mucosal cells and with the pancreatic juice and bile. Digestion, which begins in the mouth and stomach, is completed in the lumen and mucosal cells of small intestine and the products of digestion are absorbed along with most of the vitamins and fluid. The small intestine is presented with about 9 liters of fluid per day. 2 liters are from dietary sources and 7 liters from gastrointestinal secretions. However, only 1 to 2 liters pass into the colon.

Source and Composition of Intestinal Fluids					
	24 Hour Output	**Na**	**Cl**	**HCO3**	**K**
Saliva	1500 cc	9	10	15	26
Gastric Juice	2500 cc	140	150	30	5-10
Bile	500 cc	140	100	30	10
Pancreatic Juice	700 cc	140	75	75	10
Succus entericus	3000 cc	140	105	25	10

The distance from the pylorus to the ileo caecal valve in living humans is 285 cm. Throughout the length of small intestine; the mucous membrane is covered by villi. There are 20-40 villi per mm^2 of mucosa. The absorptive surface of the small intestine is increased about 600 fold by the valvulae conniventes, villi and microvilli. The inner surface area of a mucosal cylinder the size of a small intestine would be about 3300 cm^2; that the valvulae conniventes increase to 10,000 cm^2; that the villi increase to 100,000 cm^2; and that the microvilli increase to 2 million cm^2. The intestinal mucus is secreted by goblet cells in the mucosa of the small and large intestines. The mucus covers and protects the intestinal epithelium.

It also lubricates and binds some bacteria and holds immunoglobulins in place so they can bind to the pathogens. Mucous secretion is accelerated by cholinergic stimulation and by chemical and physical irritation.

There are 3 types of smooth contractions

- Peristaltic Waves
- Segmental Contractions
- Tonic Contractions

Peristaltic waves propel the intestinal contents towards the large intestine. Segmental contractions are ring-like contractions that appear at regular intervals and then disappear and are replaced by another set of ring-

like contractions in the segments between the previous contractions. They move the chyme to and fro and increase their exposure to the mucosal surface.

Tonic contractions are relatively prolonged contractions that in effect isolate one segment of intestine from another.

Very intense peristaltic waves are called peristaltic rushes and occur when the intestine is obstructed.

The intestinal glands secrete an isotonic fluid. GIT hormones such as VIP stimulate the secretion of intestinal fluid. The enzymes secreted by intestinal mucosa consist of enteropeptidase, aminopeptidase, endopeptidase and dipeptidase, maltase, lactase, trehalase and dextrinase. The hormones secreted by small intestine include secretin, GIP, VIP, neurotensin, motilin, substance P and guanylin.

Colon

The main function of colon is absorption of water, sodium and other minerals and net secretion of potassium and HCO_3. By removal of 90% of fluid it converts 1000-2000 ml of isotonic chyme that enters it each day from the ileum to about 200-250 ml of semisolid feces. Its length is about 100 cm. There are no villi in the mucosa. The movements of the colon include

segmental contractions and peristaltic waves. A third type of contraction that occurs only in the colon is the mass action contraction. These contractions move the materials from one portion of the colon to another. The first part of meal reaches the caecum in 4 hours and all of the undigested portions have entered the colon in 8 or 9 hours. The meal reaches the hepatic flexure in 6 hours, splenic flexure in 9 hours and the pelvic colon in 12 hours.

Historical perspectives

460-370 B.C	Hippocrates	Provided a detailed description of intestinal obstruction
131-201 B.C	Galen	Performed several abdominal procedures and described the anatomy of the small intestine
1670	Kerckring	Described the intestinal valvulae conniventes
1677	Peyer	Noted the presence of lymphoid follicles in the small intestine
1781-1833	Meckel	Described the diverticulum iliei verum also known as Meckel's Diverticulum

Intussusception itself has been recognized for more than three centuries:

- 1674 Paul Barbette of Amsterdam first described the intestinal invagination and suggested operative reduction
- 1751 Velse repaired an intussusception by removing the bowel, placing it in tepid milk until it returned to normal and replacing it inside the abdomen
- 1793 Hunter accurately described intussusception and discussed a post mortem specimen
- 1831 Wilson operated successfully on a Negro slave with manual reduction as reported by Thompson
- 1871 Jonathan Hutchinson performed the first successful operation for intussusception in England

- 1876 Harald Hirschsprung of Copenhagen published the first series of reports on reduction of intussusception by hydrostatic pressure. His results were superior to those achieved by operative treatment in the next 70 years.
- 1897 First successful resection of an intussusception in a child by Clubbe in Australia
- 1908 First successful resection of an intussusception in a child by Peterson in the United States
- 1913 Ladd published the first reproduction of a contrast enema radiograph showing intussusception
- 1926 Hipsley of Australia recommended the use of hydrostatic pressure from water in a rectal tube to treat intussusception
- 1926 Monrad treated intussusception using manipulative taxis through the abdominal wall after anaesthetizing his patients
- 1927 Pouliquen and de la Marnierra in France and Olsson and Pallin in Scandinavia reported their experiences with Barium enema reduction of intussusception
- 1948 Ravitch and McCune popularized the hydrostatic reduction of intussusception with barium enema.

Incidence

Intussusception occurs worldwide, but recorded geographic frequencies are strikingly variable. The relative incidence has changed little over the last 40 years. It can occur at any age. However the greatest incidence occurs in infants between 4 to 12 months of age. More than half of all cases occur within the first year and only 10-25% of the cases after the age of two. The condition has been described in premature infants and has postulated as the cause of small bowel atresia in some cases.

The absolute incidence of intussusception varies from 1.4 per 1000 live births to 1.9 per 1000 live births. Court in Newcastle upon Tyne reported more than 4 per 1000 live births[25]. Very large numbers of patients with intussusception has been reported from China with one centre in Shangai reporting 500 patients annually.

Most of the patients are well nourished healthy infants. Male versus female ratio is 3:2. This male preponderance is more striking in the 6-9 months age group[25]. Intussusception is slightly more common in white than in black children. Intussusception can occur in new borns, although only approximately 0.3% of cases occur in the first month of life. The peak

incidence of intussusception is seen in the spring and summer[25]. This peak coincides with the times of the year that gastroenteritis occurs in maximal numbers. A peak in the midwinter during the time of maximal respiratory infections has also been reported. There is also an increased incidence of intussusception among healthy infants who were recipients of Rotavirus vaccine perhaps secondary to a lead point of vaccine induced lymphoid hyperplasia.

Etiology

The cause of intussusception in most infants remains unclear. Oldham and Wesley[3] described the predisposing factors for the development of intussusception.

1. Anatomic Lead Point
    a. Meckel's Diverticulum
    b. Polyp
    c. Hypertrophied Peyer's patch
    d. Appendix
    e. Duplication or enteric cyst
    f. Lymphoma
    g. Ectopic pancreas
    h. Other neoplasms
        i. lipoma
        ii. leiomyoma
        iii. neurofibroma
        iv. metastatic tumors with or without peritoneal carcinomatosis
2. Associated infections
    a. Adenovirus
    b. Rotavirus
    c. Others
3. Bleeding disorders
    a. Henoch-Schönlein purpura

b. Hemophilia
c. Leukemia

4. Trauma
    a. Blunt abdominal trauma
    b. Major retroperitoneal operative procedures
5. Other
    a. Cystic fibrosis

Bleeding disorders and trauma are more likely to be associated with small bowel intussusception than with ileo colic intussusception[3].

In adults "two thirds rule" can be applied. Two thirds of adult intussusceptions are due to known causes. Of these, two thirds are due to neoplasms. Those caused by neoplasms, 2/3rd of neoplasms will be malignant[3].

Intussusception is composed of three parts:

- Intussusceptum – entering or inner tube
- Returning or middle tube
- Intussuscepiens – the sheath or outer tube

The part, which advances, is the apex. The mass is the intussusception and the neck is the junction of the entering layer with the mass.

An intussusception is an example of strangulating obstruction, as the blood supply of the inner layer is usually impaired. The degree of ischemia

is dependant on the tightness of invagination in which is usually greatest as it passes through the ileo caecal valve.

Types of intussusception

- Ileo colic
- Ileo ileal
- Ileo ileo colic
- Caeco colic
- Colo colic
- Jejuno jejunal
- Jejuno ileal
- Multiple
- Others

Pathogenesis

Intussusception may occur because of the disproportion between the size of the ileum and the ileocaecal valve is greater in infants than in older children. Intussusception usually originates in the distal small intestine close to the ileocaecal junction and proceeds into the ascending colon and in a much more distal direction in some instances. Intussusception higher in the small intestine may produce more severe symptoms but is much less common. Intussusception originating in the colon is uncommon in infants

but is frequent in adults. The appendix or caecum can be a lead point for intussusception.

The incidence of definite anatomic lead point ranges from 2-12% in reported series. These lead points include Meckel's diverticulum, polyp, heterotropic pancreatic nodule, enterogenous cyst, adenoma, neurofibroma, hemangioma, appendix, carcinoid tumours, foreign body, ectopic gastric mucosa or an enlarged hypertrophied ileal lymphoid patch. There are many improved theories suggesting that adenovirus or other infective agents cause primary lymphoid hyperplasia in the distal small intestine or even in mesenteric lymph nodes and that some of these enlarged lymphoid aggregates or Peyer's patches become entrapped in the intestine, serving as the lead point for intussusception. This may contribute to the development of intussusception when the diameter of the intestine is smaller than the hypertrophied lymphoid tissue. Several viruses including the reovirus and rotavirus have been incriminated in the causation of intussusception.

Intussusception has also been recorded to be a result of submucosal hemorrhage due to Henoch-Schönlein purpura, disordered coagulation, hemophilia and malignant conditions such as lymphoma, and leukemia.

Intussusception may occur after abdominal trauma and various surgical procedures. These associations with intussusception are uncommon and the disorder is most often classified as idiopathic. In idiopathic intussusception an association with prominent Peyer's patch and enlarged mesenteric lymph nodes has been observed. The most common anatomic lead point causing intussusception is Meckel's diverticulum. Undiagnosed ileoileal intussusception may be the lead point for ileo colic intussusception.

Thomas and Zachary reporting on identical twins focused attention on the occasional familial occurrence of intussusception. Simultaneous intussusception has been reported in dizygotic twins. Macmohan estimated the risk of intussusception in siblings is 1 in 40. There is a 20 times higher risk of intussusception in siblings than in a general population. The explanation for this higher risk may be due to a greater disproportion between the size of the ileum and ileocaecal valve in infants or may be due to hyperplasia and hypertrophy of peyer's patches due to viral infection[32].

In children older than 2 years of age, there is a higher frequency of specific pathologic conditions causing the intussusception. Turner,

Rickwood and Brereton reported a 22% incidence of lead points in children older than 2 years of age.

Recurrent intussusception is more common in older children than in infants. Children with cystic fibrosis are prone to secondary recurrent intussusception although spontaneous reduction may occur. It is probable that insipissated secretions and thick fecal matter in the bowel lumen acts as a lead point to produce repeated intussusception in this disease. These conditions are seen in children at an average age of 9-12 years. Diverticular disease and Celiac disease with chronically dilated flaccid bowel can also lead to intussusception in adults. Patients with Acquired Immuno Deficiency Syndrome and Kaposi's sarcoma or diffuse enteritis are also at risk of developing intussusception.

Cultures taken from the serosal surface of intussuscepted intestine that has been surgically reduced frequently revealed pathogenic bacteria. This transmural migration of bacteria was postulated in 1897 by Power and was considered a major contributor to the high incidence of infection in the pre-antibiotic era. Bacterial translocation may, in part account for the common finding of high fever after reduction of intussusception, although other

factors have now been identified. Recent studies have focused on bacteremia, endotoxemia and cytokine release in the genesis of fever with intussusception. Reactive lymphocytes have also been reported.

Development of intussusception

In most cases, intussusception begins at or near the ileo caecal valve without an obvious precipitating anatomical lesion. Simultaneous interference of the patency of the alimentary canal and with the vascular supply of the intussusception occurs. As the mesentery of the proximal bowel is drawn into the distal bowel, the mesenteric vessels are compressed in between the layers of intussusception. The slight interference with the lymphatic and venous drainage that occurs almost at once, results in edema and an increase of tissue pressure. This further increases resistance to the return of venous blood. Venules and capillaries become engorged, and bloody, edematous fluid drips into the lumen. The mucosal cells swell and goblet cells discharge mucus, which mixes with the bloody transudate in the lumen and forms the currant jelly like stool. Edema increases until venous inflow is completely obstructed. As arterial blood continues to enter the area

of intussusception, tissue pressure rises until it is higher than the arterial pressure and gangrene ensues.

The outer coat of the intussusceptum (the middle layer of the intussusception) is isolated between the two sharp bends and understandably is the first to become gangrenous. Gangrene appears in this coat near the tip of the intussusception and progresses back towards the neck of the intussusception. The final stage of intussusception is necrosis and perforation. Perforation due to gangrene occurs at the neck of the intussusception where the intussusceptum enters the intussuscepiens. Very rarely the intussusception sloughs out and anastomosis occurs with normal proximal portion of intestine producing spontaneous cure, while necrosed intussusceptum is passed per rectum.

Intussusception usually occurs in an antegrade direction. Retro grade intussusceptions are rare and reported in 0.2 % of cases. Occurrence of both antegrade and retrograde intussusception is a very rare occurrence reported in jaipur in one case where retrograde intussusception acted as a lead point for antegrade intussusception in a 7 month old baby. Multiple small bowel

intussusception either antegrade or retrograde has been observed at autopsy (agonal intussusception) [26].

Clinical Features

The cardinal symptoms and signs of intussusception from various countries show close similarities in the clinical picture of intussusception although some changes over the years have been documented. The cardinal symptoms and signs include:

- Abdominal pain
- Vomiting
- Passage of blood and mucus per rectum
- A palpable abdominal mass
- Diarrhea
- Lethargy

The symptoms at the onset tend to differ somewhat from those that predominate when the infant is seen by the surgeon.

Abdominal Pain

The presentation of intussusception in a child is classical. An otherwise fit and well male child of 5-10 months of age develops sudden onset of severe colicky intermittent pain. With the spasms of pain, the infant

commonly draws the legs up to the abdomen, screams, becomes pale and may sweat. The pain is more easily appreciated in the older child who can clearly indicate its location whereas preverbal children cannot localize the pain or complain of its presence. Pain is occasionally absent with intussusception and may not occur in children who have post operative intussusception. The abdominal pain is present in 75-83% of patients.

Vomiting

Most infants vomit in the course of illness. This symptom occurs early and is non specific. Vomiting is initially a reflex. If diagnosis is delayed, it becomes secondary to the intestinal obstruction and is often bile stained. The diagnosis should be made before this stage is reached. Vomiting is usually present in 80-85% of patients, but it was the presenting sign in only 29%.

Bleeding per Rectum

In the classical presentation, infants with the intussusception pass stools with dark red mucoid clots that resemble red currant jelly. This is caused by excess mucus production from the intussuscepted intestine that mixes with blood from the congested veins in the intussuscepted intestine. The blood in the stool may initially be found only on rectal examination. Rectal bleeding was present in 50-55% of patients.

The triad of pain, vomiting and blood per rectum is only present in one-third of the patients. One child in 10 will have diarrhoea before the signs and symptoms attributable to intussusception become obvious. This is often a cause for delay in diagnosis with potentially serious consequences.

Physical Examination

The child's vital signs are usually normal early in the course of the disease. During episodes of pain hyper peristaltic rushes may be heard.

Signé de Dance (The Sign of Dance)

The classical finding on abdominal examination of infants with intussusception in between the episodes of cramping is emptiness in the right lower quadrant. This finding is due to progression of the caecum and ileocaecal portion of the intussusception into the right upper quadrant or transverse colon.

Palpable Abdominal Mass

A sausage shaped mass is felt in the right hypochondrium extending along the line of the transverse colon. The mass is often curved because it is tethered by the blood vessels and mesentery on one side.

The mass in the abdomen is not always easy to palpate unless the infant relaxes between spasms of colic. Occasionally instead of a discrete mass in the right upper abdomen, an area of tenderness deep to the rectus muscles may be noted, when the right upper quadrant of abdomen is palpated.

A palpable abdominal mass is present in 60-78% of the patients. The absence of a palpable mass does not rule out intussusception.

Rectal Examination

On rectal examination blood stained mucus or blood may be encountered. Longer the duration of symptoms, more the possibility of identifying gross or occult blood. Intussusception occasionally passes to the descending colon or to the rectum where it may be felt on rectal examination on rare occasions. Palpation of the intussuscepted mass on bimanual examination is possible. Actual contact with the rectal examining finger is rare.

Prolapse of intussusceptum may occur through the anus. It is a grave sign, particularly when the intussusceptum is blue-black[24]. Certainly rectal protrusion of an ileal lead point is an indication of extensive telescoping and severe compromise of blood supply and ischaemic damage to the gut. Such a patient undoubtedly exhibits signs of systemic illness. The greatest danger in

a case of prolapse of intussusceptum is that the examiner will misdiagnose the condition and reduce what is seen to be merely a small rectal prolapse. To avoid such a tragedy, a lubricated tongue blade should be passed up along the side of the protruding mass before reduction is attempted. Prolapse of a colo colic intussusception may be easily mistaken for simple rectal prolapse. If the blade can be inserted more than a centimeter or two into the anus alongside of the mass, the diagnoses of intussusception should be made. Rectal prolapse, while producing discomfort, should not be accompanied by vomiting or signs of sepsis.

Pallor and occasional diaphoresis during spasms of abdominal pain are features of infants with intussusception. Lethargy commonly supervenes, which is sufficiently frequent that it should be considered as another sign indicative of intussusception. Altered consciousness and other neurological signs such as mild seizures may occasionally occur in advanced stages.

It is important to appreciate that the classical group of symptoms and signs are often absent. Absence of these cardinal signs may be responsible for the delay in diagnosis. Diarrhoea which may be present in 10% of the patients occasionally creates diagnostic confusion and may cause delay.

Fever and leukocytosis are common, particularly in infants. The pulse does not rise significantly in the initial stages of intussusception. However

episodes of colic as well as hypovolemia from dehydration often produce tachycardia. Comparing the peripheral skin temperature with the core temperatures can assess the severity of hypovolemia.

7% of the patients had had one or more previous attacks of abdominal pain in the 10 days to 6 months before the current episode. In these children there is a greater likelihood of finding a precipitating lesion causing the intussusception than with the more common idiopathic intussusception. The possibility of a lead point, such as Meckel's diverticulum should be considered. Spontaneous reduction of intussusception may occur and can be visualised using ultrasonography.

Chronic intussusception has also been described and is more likely to occur in older children who do not have complete obstruction. Chronic intussusception is rare in infants. Recurrent episodes of intussusception are more common than chronic intussusception in this age group.

Recurrent Intussusception

Recurrent intussusception has been described in 2-20% of cases, with about one-third occurring within 1 day and the majority within 6 months of the initial episode. Recurrences usually have no defined lead point and they are less likely to occur after surgical reduction or resection. There can be

multiple recurrences in the same patient. Success rates with enema reduction after one recurrence are comparable to the first episode and are better if the child did not previously require operative reduction. Patients tend to be seen earlier when suffering from recurrent intussusception and they have fewer symptoms. Irritability and discomfort may be the only clues during the early state of recurrence.

An overriding concern in recurrent intussusception is occult malignancy. For this reason it is advisable that recurrences be treated surgically in the following instances.

- A child with more than one episode of recurrence who has had no surgery to document the absence of a lead point
- Children older than 2 years whose first episode was reduced by enema
- Children in whom a pathologic lead point is suspected

Although first recurrences are successfully reduced by enema in most cases, the success rate diminishes with multiple recurrences and most individuals require at least a surgical reduction if not resection.

Post Operative Intussusception

Intussusception occurs after operations done for a variety of conditions. Thoracic as well as abdominal operations have been followed by

latent intussusception. Because ileus and adhesive obstruction more frequently come to mind as a cause for intestinal obstruction, these intussusceptions are rarely diagnosed preoperatively. Most post operative intussusceptions occur within a month of initial procedure and an interval of about 10 days between the initial operation and development of symptoms is average. Predisposing factors include suture lines, ostomy closure sites, adhesions, long intestinal tubes, bypassed intestinal segments, sub mucosal edema, abnormal bowel motility and electrolyte imbalance. Contrast radiographic studies are often obtained for diagnosis and show a small bowel obstruction. Most post operative intussusceptions are ileo ileal and respond to operative reduction without resection.

Jejunogastric Intussusception

This is a rare but potentially lethal complication of partial gastrectomy with Billroth-II anastomosis. In this condition, a portion of the full thickness of the jejunum invaginates back into the stomach. The efferent loop alone composes about 75% of jejunogastric intussusceptions; the afferent loop alone or in combination with the efferent loop constitutes the remaining cases.

The precise etiology of jejunogastric intussusception is unclear. Widely patent gastroenteric anastomoses may favour jejunogastric intussusception by permitting the loose mucosa at the anastomotic site to prolapse into the gastric pouch during normal peristalsis. Hyperperistalsis in the small bowel after gastric surgery and excessive mobility of the jejunum (especially with anitcolic anastomoses) are also contributing factors.

Jejunal intussusception may be either acute or chronic recurrent. Acute but delayed jejunogastric intussusception can occur months or years after gastric surgery. The patient with this condition presents as an acute surgical emergency, with sudden severe colicky abdominal pain, intractable vomiting and hematemesis. These symptoms reflect incarceration of the intussusceptum, and morality rises sharply with delay in surgical decompression of the upper intestinal obstruction.

Chronic jejunogastric intussusception often produces only vague symptoms of recurrent abdominal pain relieved by vomiting. Because chronic recurrent intussusception is intermittent, gastroscopy or laparotomy may fail to demonstrate the lesion.

Retrograde jejunogastric intussusception appears radiographically as a clearly defined spherical or ovoid intraluminal-filling defect in the gastric

remnant. Contrast material may be seen outlining the jejunal folds and surrounding the intussusceptum. These folds are stretched or enlarged because of the pressure edema and appear as thin curvilinear, concentric parallel stripes or striations (coiled spring appearance).

Antegrade gastrojejunal mucosal prolapse or intussusception occurs more frequently than the retrograde process. It produces a sharply marginated, smooth, occasionally scalloped intraluminal mass in the efferent or afferent loop. External compression or jejunal peristalsis may alter the size and shape of this soft flexible lesion. A large gastrojejunal mucosal prolapse may cause partial obstruction, especially if the anastomotic stoma is small.

Newer reports demonstrate that some jejunogastric intussusceptions can be reduced at fluoroscopy because of the favorable direction of the pressure exerted by the barium. Glucagon-induced hypotonia may also promote reduction of intussusception.

However in most cases of jejuno gastric intussusception (JGI), surgical exploration is necessary. A viable intussusception is reduced with or without a gastrostomy and fixation of the efferent loop is done to the parietes or the ligament of Treitz to prevent a recurrence. Gangrenous

intussusception mandates a resection. An in-situ intra-gastric resection of gangrenous jejunum followed by restoration of the continuity is one of the options. Other methods include division of the stoma and dismantling the gastro jejunostomy followed by resection and reconstruction of the gastroenterostomy.

Internal Intussusception of Rectum:

The upper rectum prolapses into the middle or lower rectum without actually reaching the anal canal. This condition is called as internal prolapse or intussusception of rectum[17]. This will cause partial obstruction to defecation. Patients complain of an urgency to defecate, a feeling of rectal fullness and pelvic pain.

1. A solitary rectal ulcer is now recognized as the cause of this syndrome
    a. The ulcer is usually located in the anterior wall in 60% of the patients. In 32% of patients it is annular and in 8%, it is posterior. Biopsies reveal a bland non neoplastic ulcer
    b. Colitis cystica profunda – which is characterized by glandular tissue beneath the mucosa, may accompany this condition. It is important to distinguish this benign lesion from cancer.

2. Internal intussusception is accompanied by abnormal rectal fixation, which permits the rectum to descend towards the perineum.

Medical treatment suffices for most patients and consists of increased dietary fiber, stool softeners, glycerin suppositories or small enemas.

Indications for surgical treatment include:

1. Debilitating symptoms despite maximum medical therapy and psychological counseling.
2. Impending anal incontinence due to stretch injury to the pudendal nerves caused by constant straining and subsequent perineal descent.
3. Chronic bleeding from a solitary rectal ulcer

Surgical treatment includes low anterior resection of the sigmoid and proximal rectum with colorectal anastomosis and rectal fixation.

Diagnosis

The clinical history may strongly suggest the diagnosis of intussusception, which is commonly confirmed on clinical examination. The investigations useful in the diagnosis of intussusception includes

1. Abdominal radiographs
2. Ultrasonography
3. Barium enema
4. CT Scan

Plain Abdominal Radiographs

In about half of the cases, the diagnosis of intussusception can be suspected on plain abdominal radiographs. Suggestive abnormalities on a plain film include:

- An abdominal mass
- Crescent of gas capping the intussusceptum, outlining its leading edge
- Target sign consisting of two concentric radiolucent curvilinear lines outlining the intussusception
- Abnormal distribution of gas and fecal contents
- Sparse large bowel gas
- Air fluid levels in the presence of bowel obstruction

- Absent caecal gas shadow in ileo ileal or ileo colic intussusception

These signs are not diagnostic, but may be useful to suspect intussusception.

Ultrasonography

Ultrasonography of the abdomen has become the standard non-invasive diagnostic technique. The sonographic pattern of intussusception was first reported in 1977. The characteristic findings in ultrasonography include

- Doughnut Sign
- Target sign or Bull's Eye lesion
- Pseudo kidney Sign

Doughnut Sign

A sausage shaped invagination is seen in USG. There is a central echogenic area representing the mucosa of the intussusceptum and a larger sonolucent rim representing the edematous wall of intussusceptum.

Target sign (Bull's Eye lesion)

If there is fluid in the lumen of intussusceptum the central core may appear sonolucent, with the next layer being the echogenic mucosa. Depending on the edema and compression of the bowel wall the layers of the

intussusception may also be seen producing the appearance of multiple concentric rings of alternating sonolucency and echogenecity. This ring will be 8 mm in thickness and with an overall diameter of more than 3 cm.

Pseudo kidney Sign

The pseudo kidney sign is seen on longitudinal section and appears as a super imposed hypoechoeic and hyperechoeic layers. This pattern represents the edematous walls of the intussusception. Successful reduction results in a smaller "donut" with an echogenic rim representing edema of the terminal ileum and ileocaecal valve.

A virtue of USG is that it minimizes the exposure to the ionizing radiation. This can be supplemented with color Doppler imaging.

Barium or Air Contrast Enema

If the doubt remains after USG, the diagnosis can be confirmed by radiography with barium or air insufflation. These procedures are usually continued from a diagnostic exercise into a treatment modality. The characteristic signs in Barium enema include

- Claw sign
- Coiled spring sign
- Bird's beak sign

Claw sign

The barium in the intussusception is seen as a claw around a negative shadow of the intussusception. This sign is present in ileo colic or colo colic intussusception. But it would be negative for the ileo ileal variant in the presence of a competent ileocaecal valve.

Coiled Spring Sign

Streaks of barium can extend around the mass in a spiral ring like fashion to become trapped between the intussusceptum and the surrounding portions of the bowel and produce the characteristic coiled spring appearance.

Intussusception of the appendix produces a oval, round, or finger like filling defect projecting from the medial wall of the caecum. The appendix is not visible. A coiled spring appearance in the caecum associated with non-filling of the appendix has been suggested as characteristic of appendiceal intussusception.

Other findings include a narrow channel of barium representing the compressed lumen of intussusceptum, a soft tissue mass on either side of this channel due to hypertrophy and oedema of the walls of the intussusceptum and intussuscipiens, and a mass lesion at the distal end of the narrow channel.

Bird's beak sign

In some cases barium studies may show proximal intestinal dilatation and a bird's beak appearance at the site of obstruction.

In equivocal cases, CT scan may further evaluate the intussusception

CT scan

Findings on CT scan may include those of a small bowel obstruction, with dilated fluid or air filled proximal loops and non-dilated loops distal to the intussusception. CT scan also reveals the presence of a small bowel mass.

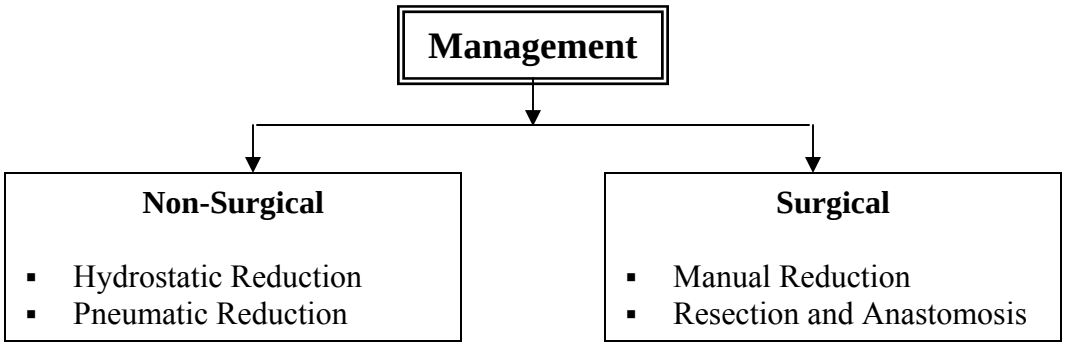
The intussusception may appear as locally distended segments of bowel with a crescent-target mass appearance from the low density mesenteric fat surrounding the intussusception. When mesenteric fat is seen three individual layers of the bowel wall can be identified; in portions of the intussusception where mesenteric fat is not present, only two layers are visible. Because of the asymmetric location of the invaginated mesenteric fat, the lumen of the intussusceptum often is eccentrically positioned.

However the availability of simpler, reliable methods makes this technique unnecessary in most infants and children. Also, the technique is no more accurate than the later approaches.

Management

It is of utmost importance in the treatment of young children with intussusception to provide intravenous fluid for rehydration, nasogastric suction and antibiotics as is done in any situation where the vascular supply to the intestine may be jeopardized. Ampicillin and Gentamicin is useful in infants. Ampicillin, Gentamicin and Clindamycin is sufficient for children older than 12 months. Adequacy of the intravascular volume can be determined by measuring the differences between core and peripheral temperatures, which should be maintained at less than 3˚C. This simple measurement is useful in judging the adequacy of fluid resuscitation.

Management of infants with intussusception is initially non-surgical. Surgery is advised only where non-surgical therapy fails. Enhancement of non-surgical reduction with administration of glucagons has been reported, as has the manipulation through the abdominal wall, with the patient under general anesthesia. However, neither technique has been widely accepted.



Non-Surgical Treatment

Hydrostatic or pneumatic reduction should be attempted only under controlled conditions. Evidence of peritonitis, perforation, advancing sepsis and possible gangrenous bowel precludes enema or pneumatic reduction. This should be the surgeon's decision. The longer the history of the symptoms the greater the possibility that enema reduction will not be successful and the more dangerous it may be.

Hydrostatic Reduction

Hydrostatic reduction of intussusception was first used successfully by Hirschsprung. By 1926 Hipsley reported 5% mortality rate with hydrostatic reduction using normal saline compared with 8% mortality rate with surgical reduction. By 1946, Barium enema reduction became the universally preferred method of treatment because of its low morbidity. Reduction of intussusception can now be visualized with ultrasonography during hydrostatic reduction.

Technique of Barium Reduction

The patient should be well hydrated. A lubricated straight catheter or Foley's catheter is inserted into the rectum and held in place by firmly taping the buttocks together. Balloon occlusion of anus must never be used. The child is restrained. A commonly used technique is the "Rule of threes". The

barium is allowed to run into the rectum from a height of 3 feet above the patient. Three attempts at reduction are made for 3 minutes each[31]. Mild sedation may be used during the procedure, but anesthesia is not recommended. A surgeon should be present when the radiologist attempts the reduction and if perforation occurs surgery should follow immediately.

Palpation of the abdomen increases the intraabdominal pressure in an uncontrolled fashion and should not be performed. When the contrast column meets the head of intussusceptum a concave filling defect occurs. This frequently presents a curvilinear spiral pattern that resembles the coil of a bedspring. The appearance is produced by the steady puddling of the barium in the compressed lumen between the head and sleeve of invagination and is not present until the barium mixture has worked itself into the folds between the intussusceptum and the intussuscepiens. The concavity flattens and the barium column moves unevenly towards the ileocaecal valve. When contrast flows freely through the valve reduction of the colonic portion of the intussusception is usually complete. The odematous valve, which can simulate an incomplete reduction of the intussusception or the lead point, may remain swollen for several days. USG can confirm that the filling defect is caused by the edematous margins of the ileocaecal valve. In ileoileocolic intussusception, the ileocolic component

may be reduced completely, with persistence of the ileo ileal lesions in the small bowel. The end point of reduction is flooding of the small bowel with the contrast material to prove the ileocolic intussusception has been reduced and to reduce an ileo ileal intussusception that may have acted as a lead point. Constant hydrostatic pressure is continued as long as the reduction is occurring. In the absence of progress, the barium is allowed to drain. This procedure can be repeated a second or a third time.

Reduction is considered complete when barium is observed as refluxing freely into the small intestine for more than 5 to 10 cm. The procedure of hydrostatic reduction may take less than 30 seconds or more than 40 minutes in more advanced cases. Confirmation of complete reduction can be obtained with ultrasonography.

Hydrostatic reduction with barium is replaced with saline, Hartman's solution or water-soluble isotonic contrast owing to the risk of Barium extravasation with perforation.

Pneumatic reduction

Adoption of the air enema or pneumatic technique has been more widespread in the late 1980's owing to higher rates of successful reduction; the procedure is fluoroscopically monitored as air is insufflated into the

rectum. The maximum safe air pressure is 80 mm Hg for young infants and 110-120 mm Hg for older infants.

Technique of pneumatic reduction

A simple manual device performs pneumatic reduction. This consists of a bulb insufflator, a sphygmomanometer, a 3-way stopcock and a tube connected to Foley's catheter. The insufflator was connected to an 18 Fr Foley's catheter that was placed in the rectum and fixed by inflating the balloon. The balloon was inflated with room air by squeezing the hand bulb. Two buttocks are firmly tapped together with wide strips of adhesive tape. Maximum of three attempts with a maximum of 3 minutes for each attempt is allowed with about 1 – 2 minutes interval between each attempt. Successful reduction is defined as copious reflux of air into the distal ileum with disappearance of soft tissue mass[27].

Pneumatic reduction is considered to be quicker, safer, less messy and decreases exposure time to radiation. Accurate pressure measurements are possible and reduction rates are higher than with hydrostatic techniques. Attempts at hydrostatic or pneumatic reduction are continued as long as progress is evident. If patients' general condition permits, 2 or 3 attempts at reduction should be done in 2 to 4 hour intervals before the procedure is abandoned. Attempts greater than 4 minutes in those patients with illness of

more than 1 day will not have any benefit, but might increase the complication rate. Success rates of reduction using hydrostatic techniques vary between 50 to 78% whereas it is 75 to 94% with pneumatic reduction. The administration of glucagons is no longer thought to be helpful as an aid in the reduction of intussusception.

Currently the method tends to be less strict. The number and duration of attempts vary according to the clinical status of the child before and after the procedure. A second trial of hydrostatic or air reduction may be undertaken within a few hours of the child does not have an acute abdomen and the symptoms relieved but the original reduction failed to show reflux into the terminal ileum. If reduction has occurred up to the edematous ileocaecal junction, watchful waiting is allowed with repeat study reserved for those with recurrent symptoms.

After reduction the patient often experiences prompt clinical improvement and falls into a deep natural sleep. After successful reduction the child is observed for 24 hours on intravenous fluids with nothing being given by mouth, The next morning antibiotics are stopped, feeding are resumed. The family should be advised of the possibility of recurrence, which exists regardless of whether the intussusception is reduced by enema or by operation.

Advantages of non-surgical treatment:

- Decreased morbidity
- Decreased cost
- Decreased length of hospital stay

Disadvantages of non-surgical treatment:

- Colonic perforation – has an incidence rate of 1 in 250 to 300 enema reductions. The causes of perforation include
    - Excessive intraluminal pressure
    - Presence of necrotic bowel
    - Existing occult perforation
- Development of Tension pneumothorax
- Poor visualization of lead points
- Relatively poor visualization of the intussusception and reduction process resulting in false positive reductions
- Recurrence is 8-12%

Contraindications

Absolute contraindications:

- Evidence of peritonitis – indicating the presence of gangrenous intestine
- Perforation

- Advancing sepsis

Relative contraindications:

- Prolonged history (> 48 hours)
- Abdominal tenderness (especially rebound tenderness)
- Small bowel obstruction
- Severe dehydration
- Profound lethargy
- High fever
- Leukocytosis

Surgical Treatment

The operative management of patients with reducible intussusception has changed greatly during the past century. Staged procedures such as Mikulicz procedure that was initially recommended by Gross, or resection with double barrel enterostomy were used during the early era of management when primary resection and reanastomosis in infants with intussusception and edematous intestine resulted in high mortality. In Glasgow, for a time a side-to-side bypass procedure was considered the best method of reducing operative time and relieving the obstruction. During the past four decades, with better management of fluid and electrolytes, improved preoperative resuscitation and safer anesthesia nearly all patients requiring resection have had successful reconstitution of the alimentary tract by end to end anastomosis.

Indications

- Signs of shock
- Signs of peritonitis
- Incomplete or failed hydrostatic or pneumatic reduction
- A residual intraluminal filling defect following enema reduction with terminal ileal reflux

Preoperative preparation

- Naso-gastric decompression
- Intravenous rehydration
- Prophylactic antibiotics

Incision

- Transverse skin incision in the right lower quadrant or immediately above the umbilicus is useful in infants
- Midline incision for adults

1. Manual Reduction

During manual reduction gentle manipulation of the bowel is needed. The intussusceptum should be gently pushed back from its distal end and not pulled out from the proximal end. Reduction is often easily achieved as far as the ascending colon, but further reduction may be difficult. In majority of cases reduction is achieved by squeezing the apex with gentle continuous pressure. Saline solution or mineral oil injected between the coats may help the reduction. If the serosa splits during this process it should be repaired before the intestine is returned to the abdominal cavity. After reduction the terminal part of the small bowel and the appendix will be seen to be reddened and stiffened with edema. The viability of bowel should be

checked carefully. If the viability of the bowel is doubtful, the application of warm saline packs may improve the circulation and relieve doubt about the necessity of resection. When serious vascular impairment has occurred, resection is usually the safest course.

Manual reduction of intussusception in adults is usually not recommended because manipulation can lead to intraluminal or intravenous tumour seeding. Colonic intussusception is never treated with manual reduction because of the extremely high likelihood of malignancy.

2. Cope's Method

In difficult cases of manual reduction, the little finger may be gently inserted into the neck of the intussusception to try and separate adhesions. Subsequently the thumb and forefinger are placed in such a way as to deinvaginate the apex[5]. Gentle pressure is applied and gradually increased to reduce the edema around the ileocaecal valve. After reduction the underlying cause requires appropriate treatment.

3. Resection and Anastomosis

Indications

- Presence of an irreducible intussusception
- Gangrenous intussusception
- Presence of a pathologic lead point

In the presence of an irreducible or gangrenous intussusception the mass should be excised in situ and the intestine is reconstituted with an end-to-end anastomosis or temporary end stoma is created. If the pathologic lead point is recognized, then resection is performed.

The involved intestine may be quite discolored from congestion, edema and hemorrhage into the wall. If the intestine is reduced, but seems to be of doubtful viability, it should be returned to the abdominal cavity for 15 minutes before it is inspected again. Reinspection may reveal a substantial improvement in the discolored intestine, but a decision to leave it or to resect it must be made. Intestine that would be considered for mandatory resection in adults is often viable in infants who have had intussusception; careful post operative surveillance is necessary if resection is not performed because perforation of the intestine may occur within a few days. The decision regarding whether to remove the appendix at the time of laparotomy is an arbitrary one for which there is little evidence to justify removal or leaving in situ, although most surgeons remove it.

Feedings are usually started the day after surgery, or when postoperative ileus shows signs of resolving, and rapidly advance to a regular diet. Antibiotics are commonly discontinued after 48 hours and the patient is often discharged shortly thereafter.

Laparoscopy

Laparoscopy is used initially in the diagnostic evaluation of hydrostatic reduction. If there is a failure of reduction laparoscopy assisted saline reduction can be performed. The use of laparoscopy definitely reduces the risk of unnecessary Laparotomy. Laparoscopy can be added safely in the standard treatment protocol for the management of intussusception[30]. The use of laparoscopy will reduce the incidence of morbidity.

Results

Patients treated by air insufflation which is simple and safe when the pressure is controlled, have a slightly higher rate of successful reduction than those treated by hydrostatic barium enema. The recurrence rate with hydrostatic barium reduction or air insufflation is higher than the 3% rate associated with surgical reduction23. The recurrence rate varies from 5-7% after hydrostatic or pneumatic reduction. Recurrence rate is 0% in a case of resection and anastomosis of the small bowel[23]. Further improvement in management has been achieved with specific quality assurance programs. In Glasgow, the resection rate during surgery has risen to more than 15% because of correction of easily reduced intussusception by non-surgical means. Perforation rate ranges from 0-3%.

Lead points that cause intussusception are rare in children younger than 2 years of age. These areas can occasionally be identified by ultrasonographic examination after air insufflation or hydrostatic reduction of the intussusception or during careful examination of the intestine at the time of reduction. Patients with a predisposing lesion should have a surgical reduction and when the inciting lesion is identified, resection should be done. The most common condition found is a Meckel's diverticulum, but various other disorders may cause intussusception.

Complications

- Hyperpyrexia due to bacteremia
- Electrolyte imbalance
- Septicemia
- Postoperative adhesions and intestinal obstruction
- Wound infection

Long term morbidity from adhesions developing after surgical reduction of intussusception has been reported. In Glasgow, a child presented acutely with gangrene of one third of small intestine resulting from an internal hernia caused by a very small adhesive band 4 years after surgical reduction of intussusception.

Mortality

The current mortality rate in children with intussusception in developed countries is less than 1%. The mortality in intussusception may be due to peritonitis secondary to gangrene or due to small bowel obstruction. The interval between the onset of symptoms and the institution of treatment is of paramount importance and the mortality rates will more nearly approach zero the more frequently treatment is instituted within 24 hours of onset. The death rate could be reduced in more than 60% of children by

eliminating the avoidable factors such as delay in diagnosis, inadequate intravenous fluid and antibiotic therapy, delay in recognizing recurrent or residual intussusception after non-surgical reduction and surgical complications.

PICTURES

Fig.1 Ileo-ileal intussusception

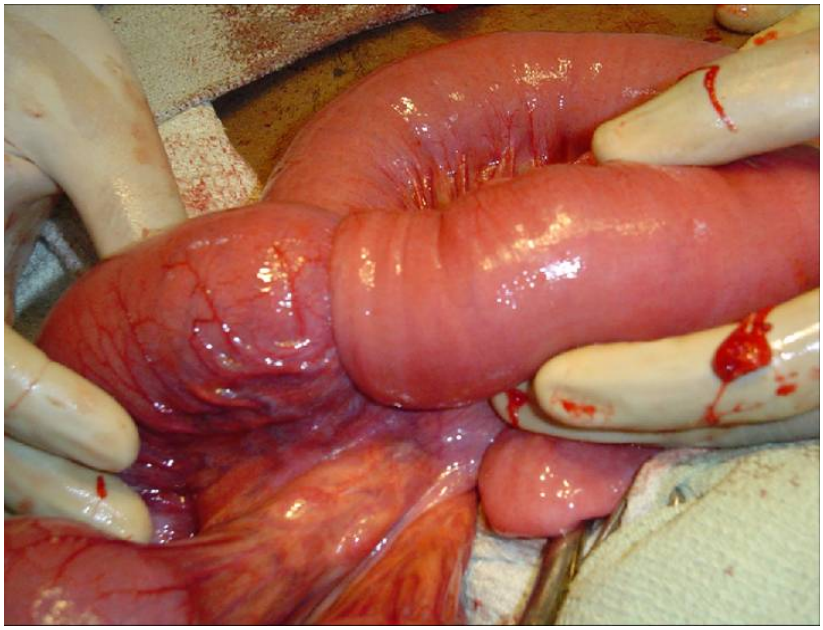


Fig.2 Meckel's diverticulum with intussusception

Fig.3 Ileo-ileal intussusception with inflammatory fibroid polyp

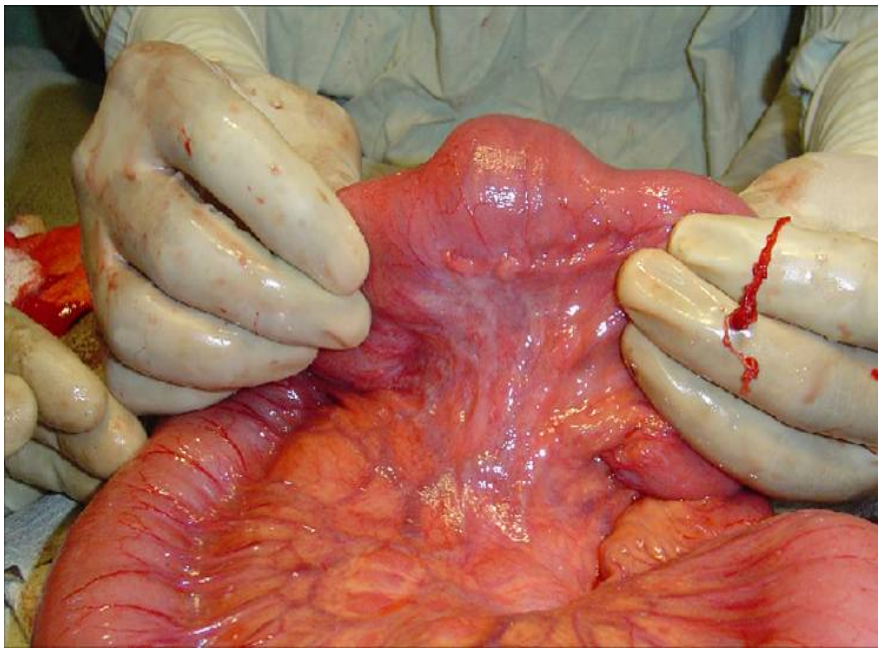


Fig.4 Inflammatory fibroid polyp

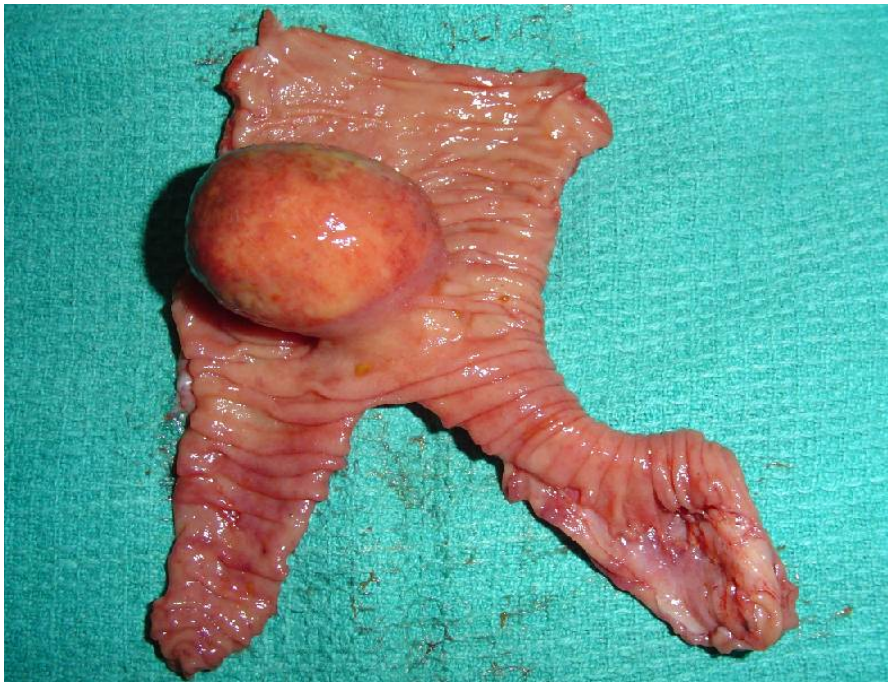


Fig.5 Ultrasound showing Target sign

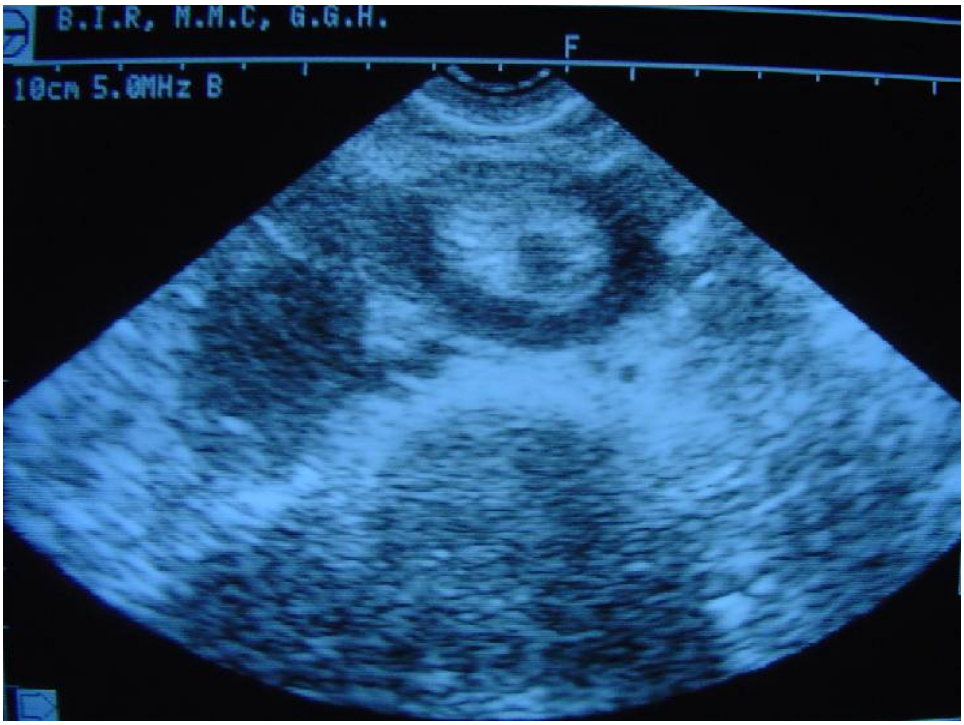


Fig.6 Barium enema – Coiled spring appearance

Fig.7 Ileal resection with end to end anastomosis

Fig.8 Meckel's diverticulum with intussusception

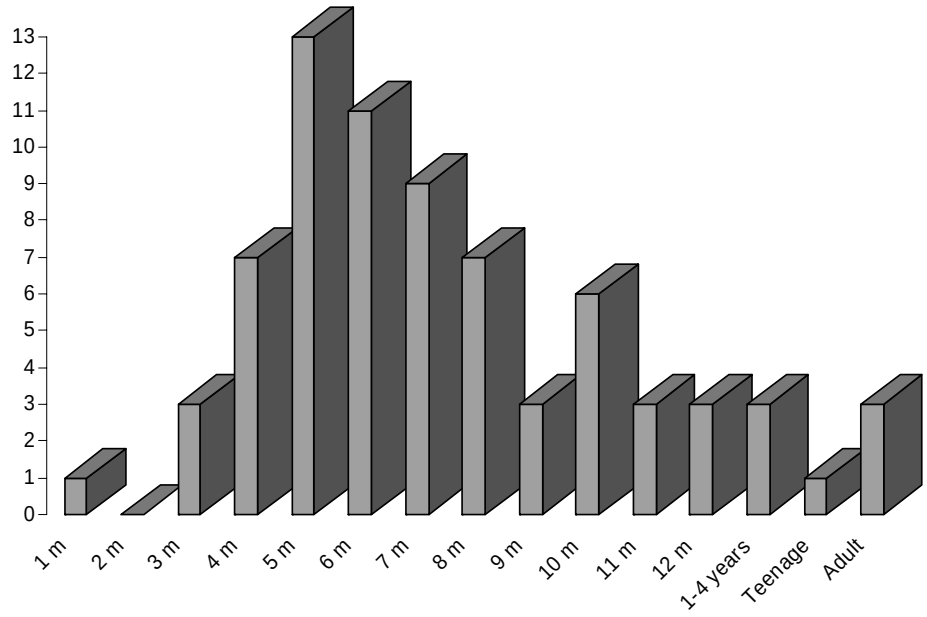
Results and Observation

Total number of cases studied: 73

Period of Study: January 2004 to December 2004

Age Distribution

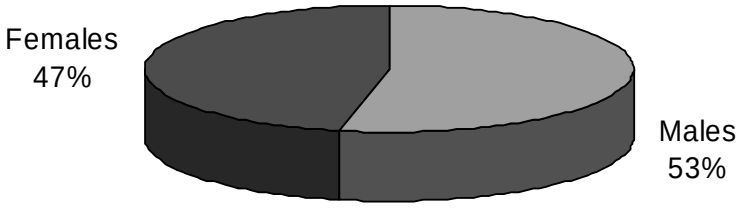
Number	Mean Age	Standard Deviation
73	1 year, 10 months	61 months



Youngest age of presentation is one month. Maximum number of intussusception occurs in the 4-8 month age group. Peak age of presentation is 5th month.

Sex Distribution

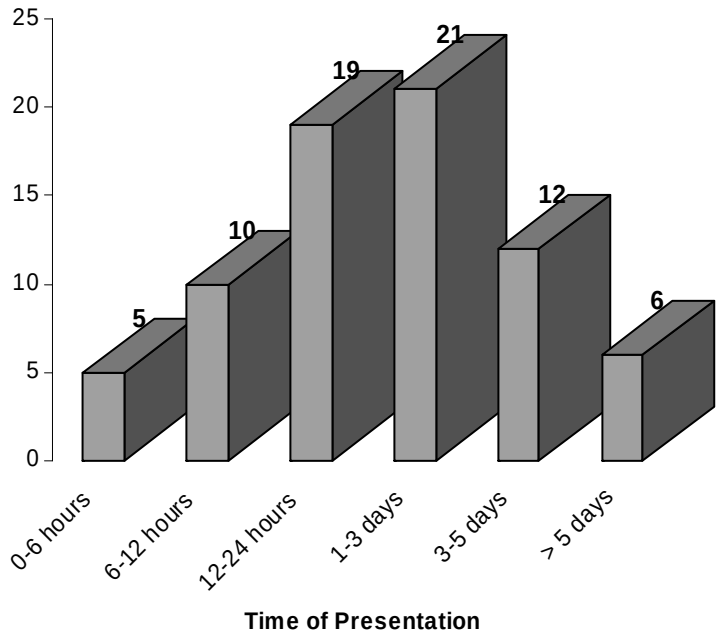
Total	Males	Females
73	39	34



There is no obvious sex difference noted in this study

Time of Presentation

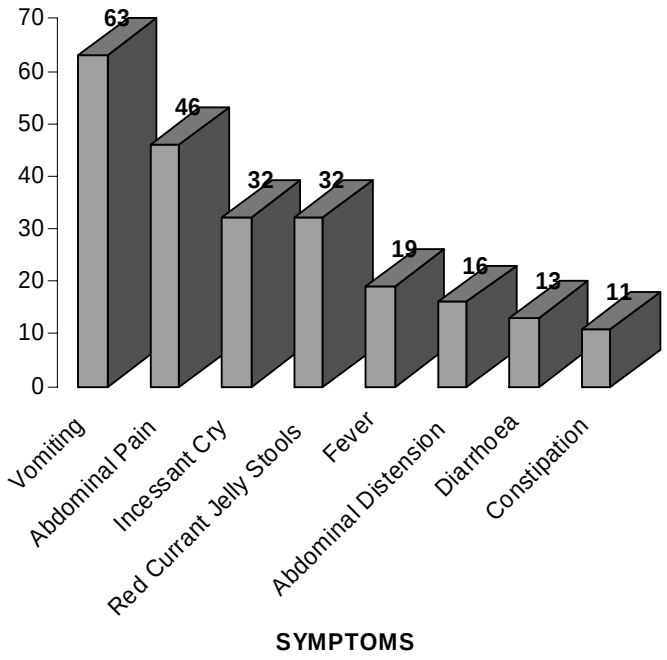
Time of Presentation	Number of Cases	Percentage
0 - 6 hours	5	7%
6 - 12 hours	10	14%
12 - 24 hours	19	26%
1 - 3 days	21	29%
3 - 5 days	12	16%
> 5 days	6	8%



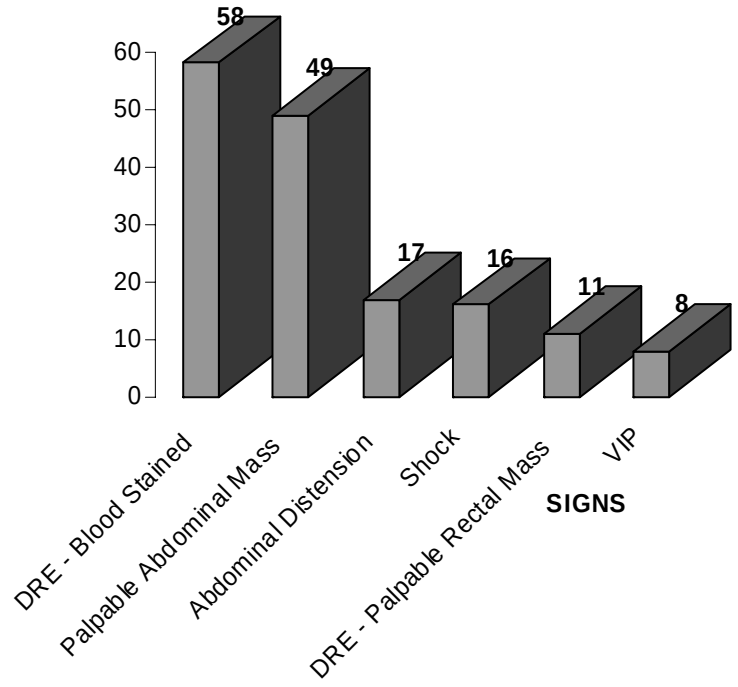
Majority of the cases present within the first 3 days of onset of intussusception.

Clinical Presentation

Clinical Symptoms	Number of Cases	Percentage
Vomiting	63	86%
Non Bilious	29	40%
Bilious	34	46%
Abdominal Pain	46	63%
Incessant Cry	32	44%
Red Currant Jelly Stools	32	44%
Fever	19	26%
Abdominal Distension	16	22%
Diarrhoea	13	18%
Constipation	11	15%



Clinical Signs	Number of Cases	Percentage
DRE - Blood Stained	58	79%
Palpable Abdominal Mass	49	67%
Abdominal Distension	17	23%
Shock	16	22%
DRE - Palpable Rectal Mass	11	15%
VIP	8	11%



Most common clinical presentation is vomiting. It is present in 86% of the cases. The classical presentation of abdominal pain, red currant jelly stools and palpable mass is seen in only 33% of the cases.

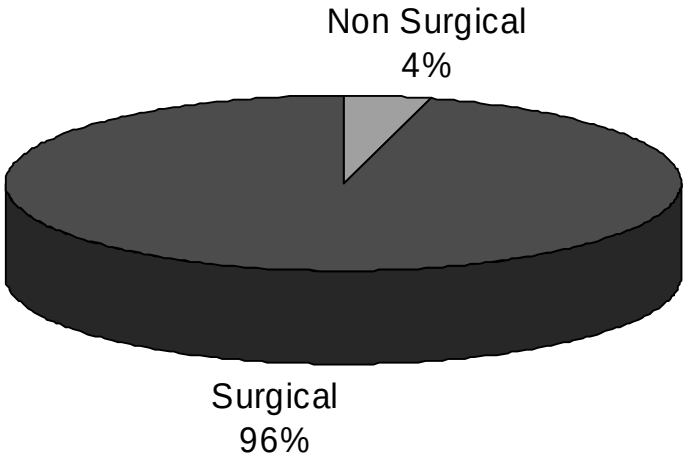
The common clinical signs to suspect intussusception are the presence of palpable abdominal mass and blood staining finger on digital rectal examination.

Investigations

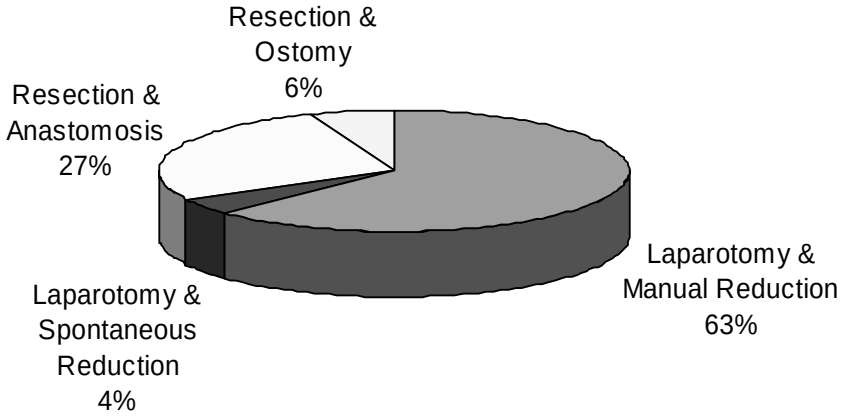
X rays are useful in 19 cases of intussusception. X rays suggestive of small bowel obstruction are seen in 21 cases. Ultrasonography is diagnostic in 14 cases.

Management

Treatment	Number of Cases	Percentage
Non Surgical	3	4%
Surgical	70	96%

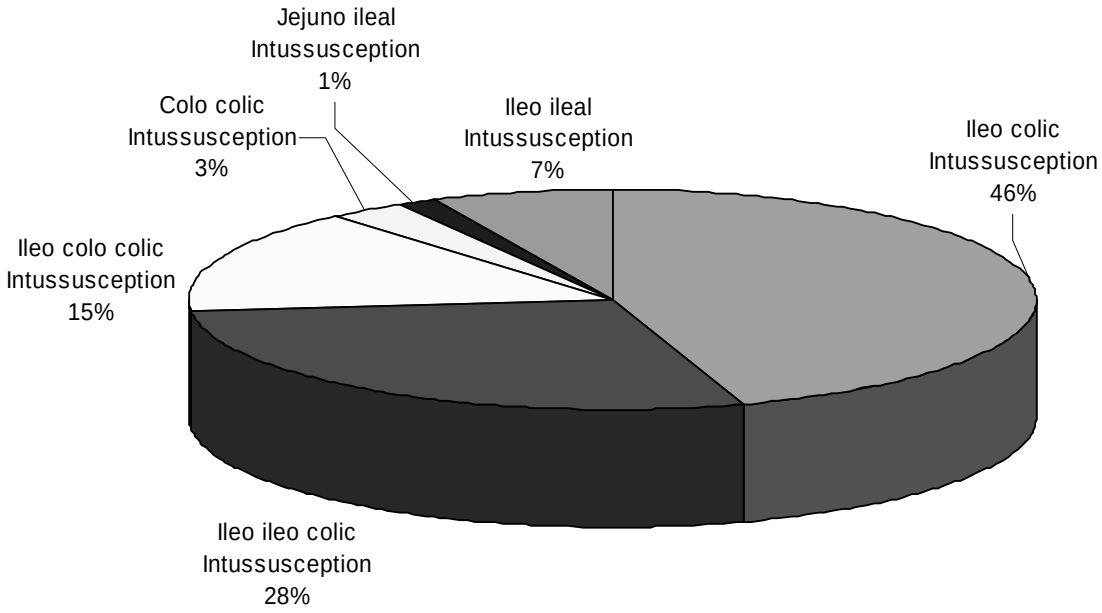


Surgical Treatment	Number of Cases	Percentage
Laparotomy & Manual Reduction	44	60%
Laparotomy & Spontaneous Reduction	3	4%
Resection & Anastomosis	19	26%
Resection & Ostomy	4	5%



Type of Intussusception

Type of Intussusception	Number of Cases	Percentage
Ileo colic Intussusception	32	44%
Ileo ileo colic Intussusception	20	27%
Ileo colo colic Intussusception	11	15%
Colo Colic Intussusception	2	3%
Jejuno ileal Intussusception	1	1%
Ileo ileal Intussusception	5	7%



Most common type of intussusception is the ileocolic intussusception. This is comparable to international statistics.

Intraoperative Finding

Intraoperative Finding	Number of Cases	Percentage
Mesenteric Nodes	8	11%
Lead Point		
Meckel's Diverticulum	5	7%
Appendix	2	3%
Polyp	4	5%

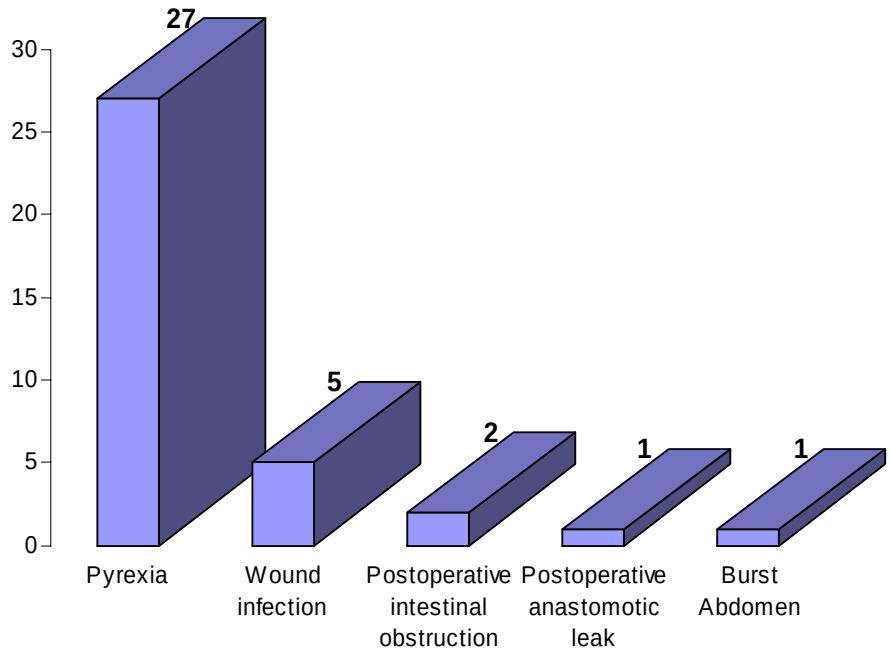
Lead point was present in 3 adult and 8 paediatric cases. Bowel perforation is noted in 3 cases. During reduction of intussusception serosal tear occurs in 4 cases.

Histopathological examination

All polyps were submitted for HPE report. One polyp was reported as inflammatory fibroid polyp and one as sub mucous lipoma and others were nonspecific. One case of Meckel's diverticulum contains heterotopic gastric epithelium.

Postoperative Complication

Complication	**Number of Cases**	**Percentage**
Pyrexia	27	37%
Wound infection	5	7%
Postoperative intestinal obstruction	2	3%
Postoperative anastomotic leak	1	1%
Burst Abdomen	1	1%



Hospital Stay

Length of Stay	Number of Cases	Percentage
< 5 days	39	53%
6 - 10 days	18	25%
> 10 days	15	21%

Mortality

Two deaths reported in our study .one death due to associated congenital heart disease and another death due to sepsis with encephalopathy. Mortality rate in our study was 2.7 %.

Discussion

Intussusception is more common in children than in adults. Intussusception occurred in 94.5% of paediatric population and in 5.5% of adult population. There is no sex predilection in our study comparable to the literature.

The peak age of incidence of intussusception in our study is 5 months of age. In the adult age group 4 cases were presented. Among the 4 adult cases, 3 cases presented with the lead point. All the 4 cases had undergone resection of the bowel. Lead point was present in 12% of paediatric cases and in 75% of adult patients comparable to the statistics. Mesenteric nodes were present in 11% of paediatric patients.

Those cases presented very late after the onset of intussusception; the complication rate is also increased. Those cases presented in a state of shock or dehydration, hydrostatic or pneumatic reduction is not possible. Also, in patients who were presented very late to the hospital, the rate of bowel resection increases and there is an increase in the rate of post operative complications.

As per literature, palpable rectal mass is a grave sign of intussusception. In our study, in 11 cases palpable rectal mass is present. Of these 11 patients, 1 was treated with saline reduction, 2 with laparotomy and

manual reduction, 8 cases underwent resection of bowel where, primary anastomosis was possible in only 3 cases. 5 cases have undergone resection and either colostomy or ileostomy. The incidence of post operative complications like wound infection and burst abdomen is more in these patients.

One case of postoperative intussusception occurs in an 11 months old infant, following Morgagni hernia repair on the 4th postoperative day. The child was taken up for emergency laparotomy. Intraoperatively, ileo ileo colic intussusception was seen and was treated with laparotomy and manual reduction.

The earlier the presentation of intussusception better is the treatment and less is the possibility for complications.

Two cases of death have been reported in our study. One death occurs in a 3 months old baby due to associated congenital heart disease Total anomalous pulmonary venous connection complicated by infective endocarditis and congestive cardiac failure with associated renal failure. Another death occurs in a 1 month old baby a case of ileo ileo colic intussusception due to acute encephalopathy with generalized tonic clonic seizures and sepsis.

Conclusion

- Intussusception is a relatively common cause of gastrointestinal obstruction in infants and young children.
- A careful history is the mainstay of diagnosis. The history is typical in most cases. Most of the cases present with abdominal pain, vomiting, red currant jelly stools and a palpable abdominal mass.
- Abdominal radiographs and ultrasonography are the primary adjuncts to careful clinical examination.
- A high index of suspicion must be maintained in atypical cases
- Patients with intussusception are optimally managed by air insufflation or hydrostatic enema
- If these methods fail, laparotomy with reduction or with resection and anastomosis is done
- Palpable rectal mass is associated with more number of resection and anastomosis as well as higher incidence of post operative complications
- Mortality rate has steadily declined during this century to well under 1%
- Delay in diagnosis is the primary avoidable factor that contributes to mortality.

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Intussusception - Master Chart

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
1	Sami, 9/12, M	446329	01/01/04	01/01/04	05/01/04	×	✓	✓	×	Ileo ileo colic Intussusception	LAP / MR
2	Sahana, 6/12, F	446880	02/01/04	03/01/04	06/01/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
3	Amsha, 4/12, F	446999	04/01/04	04/01/04	07/01/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR Appendectomy
4	Ashik Ahmed, 5/12, M	447783	13/01/04	13/01/04	16/01/04	✓	×	×	✓	Ileo colo colic Intussusception	LAP / MR
5	Priyadharshini, 6/12, F	447824	13/01/04	13/01/04	18/01/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / MR
6	Bhuvaneshwari, 3/12, F	448114	18/01/04	18/01/04	22/01/04	×	✓	✓	✓	Ileo colo colic Intussusception	LAP / MR
7	Akash, 4/12, M	448307	20/01/04	20/01/04	23/01/04	×	✓	×	×	Jejuno ileal Intussusception	LAP / MR
8	Deepalakshmi, 5/12, F	448831	25/01/04	25/01/04	28/04/04	×	×	✓	✓	Ileo colic Intussusception	LAP / MR
9	Hemanath, 4/12, M	449903	06/02/04	06/02/04	09/02/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
10	Gokul, 1/12, M	450495	12/02/04	12/02/04	19/02/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / RA, sepsis, metabolic encephalopathy. Death
11	Keerthika, 7/12, F	450598	12/02/04	12/02/04	16/02/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
12	Satheeshwaran, 7/12, M	450694	13/02/04	13/02/04	18/02/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / MR

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
13	Jilanibaasha, 5/12, M	450701	13/02/04	14/02/04	19/02/04	✓	✓	✓	✓	Ileo colo colic Intussusception	LAP / RA
14	Rajkumar, 5/12, M	450778	14/02/04	14/02/04	20/02/04	×	✓	✓	✓	Ileo colic Intussusception	LAP / MR
15	Senthoorpondi, 6/12, M	450836	15/02/04	15/02/04	19/02/04	×	✓	✓	✓	Ileo colic Intussusception	LAP / MR
16	Keerthana, 7/12, F	450872	16/02/04	16/02/04	19/02/04	✓	✓	✓	✓	Ileo caeco colic Intussusception	LAP / MR
17	Anbarasi, 7/12, F	451019	16/02/04	16/02/04	19/02/04	✓	✓	✓	×	Caeco colic Intussusception	LAP / MR
18	Darwin, 1, M	451118	17/02/04	17/02/04	21/02/04	✓	✓	×	✓	Ileo colic Intussusception	LAP / MR
19	Karthik, 2, M	452731	04/03/04	05/03/04	13/03/04	✓	✓	×	✓	Ileo ileal Intussusception	LAP / RA
20	Mahalakshmi, 6/12, F	453619	13/03/04	13/03/04	17/03/04	×	✓	✓	×	Ileo ileo colic Intussusception	LAP / MR
21	Teja, 10/12, F	454067	18/03/04	18/03/04	24/03/04	×	✓	✓	✓	Ileo colic Intussusception	LAP / RA
22	Deen, 6/12, M	454140	18/03/04	18/03/04	22/03/04	×	✓	✓	✓	Ileo colic Intussusception	LAP / MR
23	Vigneshwari, 5/12, F	454736	24/03/04	25/03/04	28/03/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / SR
24	Sakthivel, 30, M	651340	27/03/04	27/03/04	12/04/04	✓	✓	✓	✓	Ileo ileal Intussusception	LAP / RA
25	Gayathri, 5/12, F	455303	30/03/04	31/03/04	06/04/04	×	×	✓	✓	Ileo ileo colic Intussusception	LAP / RA
26	Poovarasan, 11/12, M	455492	01/04/04	16/04/04	26/04/04	✓	✓	×	×	Post operative ileo ileo colic Intussusception	LAP / MR

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
27	Sri Krishna, 7/12, M	455592	02/04/04	02/04/04	11/04/04	×	✓	✓	×	Ileo colic Intussusception	LAP / RA
28	Ganesh, 7/12, M	456068	08/04/04	08/04/04	20/04/04	×	✓	✓	✓	Ileo colo colic Intussusception	LAP / Resection Ileostomy
29	Lakshmipriya, 8/12, F	456234	09/04/04	10/04/04	14/04/04	×	✓	×	×	Ileo colic Intussusception	LAP / MR
30	Dharshini, 5/12, F	456527	13/04/04	13/04/04	16/04/04	×	✓	✓	✓	Ileo caeco colic Intussusception	LAP / MR
31	Yuvan Raj Shankar, 6/12, M	456655	14/04/04	15/04/04	17/04/04	✓	✓	×	✓	Ileo colic Intussusception	LAP / MR
32	Indumathi, 10/12, F	456998	19/04/04	19/04/04	25/04/04	×	×	✓	✓	Ileo ileo colic Intussusception	LAP / RA
33	Sofia, 7/12, F	457861	26/04/04	26/04/04	29/04/04	✓	✓	×	✓	Ileo caecal Intussusception	LAP / MR
34	Samu, 8/12, M	458060	28/04/04	28/04/04	03/05/04	✓	✓	✓	✓	Ileo caeco colic Intussusception	LAP / MR
35	Umapathy, 8/12, M	458148	29/04/04	30/04/04	11/05/04	✓	✓	✓	×	Ileo ileo colic Intussusception with Perforation	LAP/RA
36	Suriya, 8/12, M	458310	01/05/04		04/05/04	✓	✓	✓	✓	Intussusception	Hydrostatic Reduction
37	Logesh, 7/12, M	458495	03/05/04	04/05/04	06/05/04	×	✓	✓	✓	Ileo caecal Intussusception	LAP / MR
38	Haridoss, 22, M	657690	04/05/04	04/05/04	16/05/04	✓	✓	×	×	Ileo colic Intussusception	LAP / RA
39	Dinesh, 10/12, M	458673	05/05/04	05/05/04	13/05/04	✓	✓	✓	✓	Ileo ileo colic Intussusception with Meckel's Diverticulum	LAP / RA

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
40	Ugishree, 6/12, F	458728	06/05/04	06/05/04	08/05/04	×	✓	✓	✓	Ileo ileo colic intussusception	LAP / MR
41	Sithiq, 6/12, M	459292	11/05/04	12/05/04	27/05/04	✓	✓	✓	×	Ileo ileo colic Intussusception with Perforation	LAP / RA
42	Soundari, 6/12, F	460084	19/05/04	19/05/04	24/05/04	×	✓	✓	×	Ileo ileo colic Intussusception	LAP / MR
43	Vignesh, 7/12, M	460500	22/05/04	22/05/04	26/05/04	✓	✓	✓	×	Ileo colic Intussusception	LAP / MR
44	Mithruka, 11/12, F	460746	24/05/04	24/05/04	28/05/04	✓	✓	×	✓	Ileo caeco colic Intussusception	LAP / MR
45	Padmavathy, 3.5, F	460774	24/05/04	24/05/04	28/05/04	✓	✓	×	✓	Ileo colic Intussusception	LAP / SR
46	Kirubas, 1.5, M	461239	29/05/04	29/05/04	05/06/04	✓	✓	✓	×	Ileo colic Intussusception	LAP / MR
47	Rajesh, 10/12, M	461335	30/05/04	30/05/04	10/06/04	✓	✓	✓	✓	Ileo colic Intussusception with Meckel's Diverticulum	LAP / RA
48	Kameshwaran, 8/12, M	462907	14/06/04	14/06/04	17/06/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
49	Punitha, 5/12, F	463461	20/06/04	20/06/04	07/07/04	✓	✓	×	×	Ileo colic Intussusception with perforation	LAP / Resection ostomy
50	Shagul, 10/12, M	464022	25/06/04	25/06/04	08/07/04	✓	✓	✓	×	Ileo ileo colic Intussusception	LAP / RA
51	Logesh, 5/12, M	464190	26/06/04	26/06/04	01/07/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / MR

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
52	Roshan, 3/12, M	464588	30/06/04	30/06/04	04/07/04	×	✓	✓	✓	Ileo colic Intussusception	LAP / MR
53	Keerthiga, 5/12, F	465139	05/07/04	05/07/04	09/07/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
54	Resma Thaarani, 11/12, F	467716	28/07/04	29/07/04	03/08/04	✓	✓	✓	✓	Ileo ileo colic Intussusception with Meckel's Diverticulum	LAP / RA
55	Eswari, 8/12, F	468929	09/08/04	10/08/04	12/08/04	✓	✓	✓	✓	Ileo colic Intussusception	Pneumatic Reduction, Saline Reduction, LAP / SR
56	Joswa, 9/12, M	470222	20/08/04	20/08/04	26/08/04	✓	✓	✓	✓	Ileo colo colic Intussusception	LAP / MR
57	Srikanth, 3/12, M	470906	26/08/04	09/09/04	15/09/04	✓	✓	✓	✓	Ileo ileal Intussusception with CHD, CCF and Renal failure	LAP / MR, Death
58	Yogesh, 1, M	471775	03/09/04	03/09/04	07/09/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / MR
59	Harisudhakar, 4/12, M	472238	06/09/04	06/09/04	01/10/04	✓	✓	✓	✓	Ileo ileo colic Intussusception with Meckel's Diverticulum with postoperative intestinal obstruction	LAP / RA
60	Poornima, 8/12, F	473528	19/09/04	19/09/04	22/09/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR Appendectomy
61	Anjana, 4/12, F	473688	20/09/04	21/09/04	08/10/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / Resection Ostomy

No	Personal Details: Name, Age, Sex	IP No	DoA	DoS	DoD	Pain Abdomen	Vomiting	Bleeding PR	Palpable Mass	Diagnosis	Management
62	Aneesbanu, 9/12, F	474510	28/09/04	28/09/04	06/10/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
63	Raghu, 22, M	684747	01/10/04	01/10/04	22/10/04	✓	✓	✓	✓	Colo colic Intussusception with polyp	LAP / Resection and Colostomy
64	Muthulakshmi, 13, F	685102	03/10/04	03/10/04	16/10/04	✓	✓	✓	✓	Ileo ileal Intussusception with Meckel's Diverticulum	LAP / RA
65	Balachandran, 5/12, M	475294	04/10/04	04/10/04	11/10/04	✓	✓	✓	✓	Ileo colic Intussusception with polyp	LAP / RA
66	B/o Dhanamma, 5/12, M	475574	06/10/04	06/10/04	11/10/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
67	Pavithra, 6/12, F	476796	15/10/04	16/10/04	18/10/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
68	Kayalvizhi, 10/12, F	477472	21/10/04	21/10/04	04/11/04	✓	✓	✓	✓	Ileo ileo colic Intussusception	LAP / RA
69	Pavashri, 4/12, F	478467	29/10/04	29/10/04	02/11/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
70	Madhani, 4/12, F	479238	05/11/04	05/11/05	11/11/04	✓	✓	✓	✓	Ileo ileal Intussusception	LAP / MR
71	Gokul, 1, M	481592	24/11/04	25/11/04	09/12/04	✓	✓	✓	✓	Ileo colo colic Intussusception with polyp	LAP / RA
72	Harsini, 5/12, F	482516	01/12/04	01/12/04	05/12/04	✓	✓	✓	✓	Ileo colic Intussusception	LAP / MR
73	Dhivya, 6/12, F	482547	02/12/04		05/12/04	✓	✓	✓	✓	Ileo colo colic Intussusception	Saline Reduction

LAP/ MR - LAPAROMY AND MANUAL REDUCTION
LAP / SR – LAPAROTOMY AND SPONTANEOUS REDUCTION
LAP / RA – LAPAROTOMY, RESECTION AND ANASTOMOSIS